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A REVIEW OF 268 CASES OF GASTRECTOMY FOR PEPTIC ULCER.[1]

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This review, up to 1955, includes the series of 50 cases reported previously (Kinsella, 1949). Patients in St. Vincent's Public Hospital numbered 179, in Hornsby District Hospital 47, and in private hospitals 41.

During the same period, but not included in this series, six patients had gastro-enterostomy (in addition to the 10 reported in 1949), and five had vagotomy (in addition to the three reported in 1949). No patient was refused gastrectomy on account of the difficulties at the site of ulcer.

Gastro-enterostomy has been performed much less frequently in recent years. It was performed on patients with high-grade stenosis, who had, at the same time, other complications responsible for a very poor general condition. Thus, one female patient, referred by Dr. B. Hall, was aged ninety-two years; another woman, referred by Dr. G. Hall, had recently lost a toe from gangrene, and had diabetes, coronary sclerosis and recent uræmic coma.

[1] Since this paper was submitted for publication in July, 1955, the author's series had increased up to December 25 to 280 with no further mortality.

Gastro-enterostomy should not be performed for non-obstructive duodenal ulcer, or for gastric ulcer. Figure I shows large gastric and duodenal ulcers, four years after an ineffective gastro-enterostomy.

Vagotomy was performed for six patients with small, non-penetrating, non-obstructive, but intractable duodenal ulcer, and a rapidly emptying hypertonic, hyperacid stomach; for one patient with intractable gastritis in a similar type of stomach; and for one patient with a stomal ulcer and a gastro-colic fistula and an epigastrium occupied by a broad, thick cheloid, and many other disabilities, after a recent gastrectomy and further operations, all performed overseas.

Pre-operative Complicating Diseases.

Several patients had severe hypertension, blood pressures of 260 millimetres of mercury, systolic, and 150 millimetres, diastolic, being the highest reading before treatment. Several patients had portal cirrhosis of moderate degree. One patient with a high gastric ulcer had ankylosing spondylitis, and such severe kyphosis that a resection of his costal margin and part of the sternal body was necessary to provide access to the stomach. Numerous patients had severe bronchitis—the so-called "cigarette cough". Several had hernias through previous incisions for suture of ruptured ulcer.

Five patients had extensive quiescent pulmonary tuberculosis. One man, referred by Dr. Nevell, had been off work for five years on account of bilateral disease. He is now well, and working, two years after gastrectomy.

The other patients also returned to work. One, in addition to her pulmonary disease, had Pott's disease for which a spinal graft had been performed.

One patient, a man, aged seventy-one years, referred by Dr. J. Sherwood, was admitted to the medical wards on account of severe hæmatemesis. On the seventh day he suffered an acute coronary infarction, confirmed by electrocardiographic examination. Gastrectomy was performed three days later on account of continued bleeding from a large posterior-penetrating duodenal ulcer, which could be removed only in its proximal part. He progressed well for five days, and then died suddenly from severe hæmorrhage from the remaining distal part of his ulcer. The presence of the acute antero-lateral coronary infarction was confirmed at autopsy.

One patient had a subacute cholecystitis and cholelithiasis, and cholecystostomy was performed with gastrectomy.

Age Groups.

The age groups were as follows: twenty to twenty-nine years, 11 patients; thirty to thirty-nine years, 38 patients; forty to forty-nine years, 82 patients; fifty to fifty-nine years, 83 patients; sixty to sixty-nine years, 42 patients; seventy to seventy-nine years, 12 patients.

The youngest patient was aged twenty-one years and the eldest seventy-seven years.

Type of Ulcer and Sex Distribution.

The type of ulcer and sex distribution are shown in Table I.

TABLE I.
Type of Ulcer and Sex of Patients.

Type of Ulcer.	Patients.		
	Male.	Female.	Total.
Gastric	58	48	106
Duodenal	93	16	109
Pyloric	9	6	15
Gastric and duodenal	9	6	15
Stomal	3	3	6
Gastritis (understated)	10	7	17
Total	182	86	268

Multiple ulcers in stomach or duodenum have not been separately recorded.

Gastritis has been included because it is so closely related. I failed to distinguish chronic gastritis from ulcer in my first review, so that the incidence of chronic gastritis is slightly understated in Table I. The "erosion", a mucosal breach no deeper than the *muscularis mucosæ*, should be distinguished from ulcer. It indicates a widespread chronic disease of the mucosa. Some British gastro-enterologists, misled by innocent macroscopic and gastroscopic appearances, have not yet realized this. Chronic gastritis still masquerades in the tables under "acute ulcer", "gastric bleeding, no ulcer found", "site of bleeding point unknown", "no abnormality", "neurosis", *et cetera*. The term "vicarious menstruation" has been dropped, but "gastrostaxis" still lingers.

The term "pyloric" is given to junctional ulcers, which could not be more exactly classified, apart from microscopic examination of the surrounding mucosa.

Stomal Ulcer.

There were six stomal ulcers. One of these followed an interval gastrectomy in this series (an incidence of one in 238), and this one could have been avoided by a better knowledge. Four patients had been operated on elsewhere.

CASE I.—Mrs. A. had a stomal ulcer following a small Billroth I procedure.

CASE II.—Mr. B. had a stomal ulcer following a gastroenterostomy placed so high that it functioned badly.

CASE III.—Mrs. C. had had localized excision of a lesser curvature ulcer, and later a gastro-enterostomy with badly placed stoma. She was admitted to Saint Vincent's Hospital, mentally deranged, and almost moribund from malnutrition. A stomal ulcer had completely constricted the stomach, so that not even a thin barium emulsion could leave the upper gastric compartment (Figure II).

Rydygier and Czerny are credited with the introduction of localized excision of gastric ulcers in 1881. Continental surgeons soon found that the operation gave poor results. English and American surgeons came to the same conclusion about forty years later.

CASE IV.—Mr. D. was admitted to hospital in 1942 with stomal ulcer following a gastro-enterostomy placed so high that it functioned badly. I performed a partial gastrectomy with anastomosis of afferent jejunal loop to greater curvature (a method which should be abandoned), but removed insufficient greater curvature for this type of anastomosis, and the patient again developed a stomal ulcer. After a second partial gastrectomy in 1951, he is now symptom-free.

CASE V.—Mrs. E. (discussed in the next section) had a gastrectomy, which was followed by stomal ulcer partly because of her extraordinary tendency to ulcer formation, partly on account of the method of anastomosis.

CASE VI.—Mr. F. had had a gastro-enterostomy for duodenal ulcer twenty-two years previously, with further operation twelve months later. Ten years later again his stomal ulcer became evident.

From a study of these cases, and of the literature, the conclusion emerges that the chief cause of stomal ulcer is defective surgery. Crude stitching, faulty arrangement of the stoma and loops, and insufficient removal of gastric tissue are the usual factors.

Total Gastrectomy.

CASE I.—Mrs. G., aged sixty-three years, was admitted to Saint Vincent's Hospital on October 17, 1950, and was thought to have carcinoma of the pylorus. At operation the mass found at the pylorus was thought to be carcinoma, and high on the anterior wall was a small, hard, white, outlying nodule. Total gastrectomy was performed. Microscopic examination showed the ulcer to be simple and the nodule to be a leiomyoma. She has remained symptom-free until the present time, and is under observation by Dr. W. McGrath.

CASE II.—Mrs. E., aged forty-two years, was admitted to Dorrigo Hospital on June 26, 1948, with hæmatemesis. During five weeks she was given six litres of blood and serum, but the bleeding persisted. On July 28, 1948, she was admitted to the medical ward of Saint Vincent's Hospital. During the first fifteen hours after her admission to hospital, she was given one and a half litres of blood, but her hæmoglobin value remained at 19% and her red cells numbered 900,000 per cubic millimetre. She was examined by Dr. B. Hall, who found her *in extremis*, and considered further medical treatment to be useless. On July 29, 1948, she again vomited bright blood and was unfit for operation. A Levin tube was placed in the stomach and aspirated hourly (in order to give early notice of further bleeding), and four litres of blood were given rapidly. The hæmoglobin value rose to 92% and the number of red blood cells to 4,000,000 per cubic millimetre; but aspiration of the Levin tube revealed bright blood. Immediate operation was arranged. During field block anæsthesia of the anterior abdominal wall, the patient became restless and complained of nausea, and shortly afterwards began to struggle and scream, with severe abdominal pain. Intravenous anæsthesia was now given. The abdomen was seen to be swelling rapidly and visibly and the patient's condition deteriorating. Meantime, the resident medical officers, under the direction of Dr. R. C. Craven, were not only attending to the anæsthesia, but giving three and a half litres of blood rapidly through two veins. A Murphy rectal drip administration of tap-water was also given, for patients whose blood is being replaced need also water. The abdomen was opened through a mid-line incision. The stomach was extremely distended with blood visible through the thinned wall, and on the point of rupture. The incision in the abdominal wall was extended and an incision was made in the long axis of the anterior wall of the stomach. Blood gushed out and flowed in torrents to the floor over each side of the table, and was scooped out by hand. A strong jet of blood was seen (and heard, by Dr. R. C. Condon) coming from the splenic artery, which lay exposed in the floor of a huge penetrating posterior wall gastric

ulcer. Hæmorrhage was controlled by digital pressure, and the splenic artery was underrun by a silk suture. This cut out; but two catgut sutures succeeded. The gastric ulcer extended from the lesser to the greater curvature and involved the middle third of the stomach. It had completely divided the posterior wall, so that only the anterior wall remained for division by the scalpel. In addition, there was a duodenal ulcer mass involving the liver and the head of the pancreas. An exclusion gastrectomy was performed, but the antral mucosa was not removed. The proximal third of the stomach was left firmly attached to the posterior abdominal wall, and the left gastric artery was not sought for in the mass of fibrous tissue. The upper jejunal coil was laid in the ulcer crater, sutured to the floor and proximal edge of the ulcer and opened, and the anterior lip of the incision was sutured to the anterior wall of the stomach, the anastomosis being thus completed. The steps are shown in Figures III and IV. Convalescence was slow, and she was
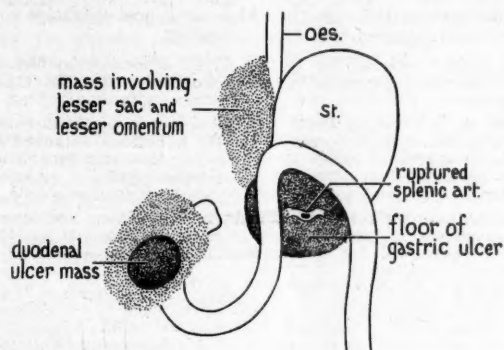

FIGURE III.

Mrs. E.: gastrectomy for gastric ulcer and ruptured splenic artery.

discharged from hospital in good condition on September 4, 1948. Figure V is the skiagram taken before her discharge from hospital; it shows the stoma working well, and the large niche of the duodenal ulcer. Several months later, symptoms recurred, and the skiagrams showed a lesser curvature stomal ulcer (Figure VI). On August 30, 1949, the remainder of the stomach was removed, a posterior trans-thoracic extrapleural approach being chosen in order to secure more direct access to the origin of the left gastric artery. The œsophagus and jejunum were anastomosed en Roux. In August, 1950, the patient was symptom-free and of normal weight.

She remained well until October, 1953, when she contracted gastric influenza, gastro-enteritis and pneumonia. She was admitted to the Mater Misericordiæ Hospital, Sydney, with acute pneumonitis. The infection was followed by a severe anæmia, which recovered under treatment. In February, 1955, she reported that her blood count, performed at Lismore, was almost normal, and that she was "feeling well, about 8st. 2 lb. [her normal weight], still with a good appetite, and eating almost like an ordinary female". The "almost" meant that she felt full after large meals.

It is of interest to note that these patients retain their appetite, and their sense of hunger, in spite of the theory of Carlson that hunger is due to powerful gastric peristalsis. His so-called "hunger-pangs" are probably due to the efforts of the outraged stomach to expel the distended balloon placed therein by the physiologist.

It is also of interest that these two patients have remained free from anæmia, except on the occasion of a febrile attack. It is probable that, in these patients, anæmia is prevented by a good diet and freedom of the duodeno-jejunal mucosa from progressive atrophic changes. Fellinger (1950) studied, for at least four years, eight patients operated on by Finsterer, and found that, with a good diet, there was sufficient intrinsic factor to maintain blood health, but with war-time malnutrition and enteritis a severe megalocytic anæmia developed, presumably through lack of extrinsic factor. Megalocytic anæmia

develops late, if at all. But slight to severe normocytic anæmia may develop earlier.

Anæsthesia.

When I began this work in 1937 I used local anæsthesia, sometimes supplemented by ether, nitrous oxide and oxygen or "Pentothal". But more recently, when general anæsthesia improved, and anæsthetists trained in the new methods became available, I adopted general anæsthesia. I therefore felt a little shame-faced when I found Barclay and McIntyre (1954), in New Zealand, still stoutly affirm-
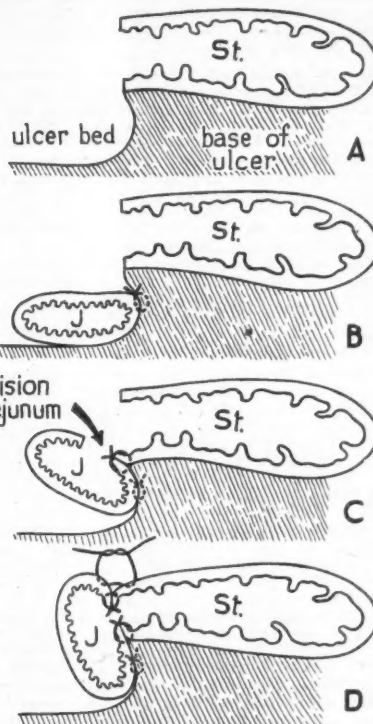
FIGURE IV.

Mrs. E.: steps of anastomosis.

ing that "the use of local anæsthesia alone has become standard practice and gives the utmost satisfaction. During and since the period under review, use of the most modern and expert general anæsthesia has repeatedly confirmed my (S.B.'s) belief in the overwhelming superiority of local anæsthesia alone". They report good results in 90 cases.

In earlier years, intravenous infusion of Ringer's solution was begun before operation (in order to obviate the delays when an inexpert anæsthetist was required to give an intravenous supplement), and continued after operation. In recent years it has been given up, for reasons stated later.

Mortality.

Among the 242 interval cases (including eight cases in which a previous gastro-enterostomy had been performed), there have been four deaths, a mortality of 1·6%. This is rather disappointing, in view of the zero mortality reported in 1949.

However, since March, 1950, there have been 161 consecutive "interval" cases without a death.

The four deaths in the interval cases were as follows:

Mr. I., aged forty-three years, was admitted to Saint Vincent's Hospital on November 18, 1948, with a large

penetrating duodenal ulcer, and a very large incisional hernia following three previous operations, one for ruptured ulcer, the second an attempted operation on the stomach, and the third an attempted repair of the incisional hernia. His pain was exceptionally severe. He was undernourished, and it was found later that he was addicted to methylated spirits. He had a curious bronzed complexion which was at first thought to be due to vitamin deficiency; but it did not improve during two months of pre-operative medical treatment in hospital. After operation on January 18, 1949, he was non-cooperative, repeatedly pulled out the intravenous needle and intranasal tubing and had tachycardia. On the morning of January 21, 1949, he sat up in bed and complained of pain in the upper sternal region; his pulse rate rose, his blood pressure fell, and he died on that same afternoon. Clinically, the cause of death was thought to be coronary insufficiency. Autopsy did not reveal the cause of death. The operation area was normal.

Mr. J., aged seventy-three years, had asthma for forty years, a posterior duodenal ulcer penetrating the pancreas, an anterior duodenal ulcer adherent to the liver, and severe portal cirrhosis with obvious distension of the splanchnic veins. After operation (on February 22, 1949), bleeding from the wound occurred and the blood remained unclotted, and purpura appeared on the back of his hands. The prothrombin time was 50% of normal. Bleeding stopped on February 25, 1949; the patient appeared well till midnight of February 26. He then had a rigor, his temperature rose to 103° F. and he became comatose with feeble pulse, and he died at noon on February 27, 1949. Autopsy revealed a calcified and completely adherent pericardium, and a very thin-walled right ventricle. The operation area was normal, and there was no blood in stomach or intestines. The autopsy did not reveal the cause of death. The cranial cavity was not opened.

Mr. K., aged forty-eight years, was referred by Dr. O. A. Diethelm on account of repeated severe melæna. Operation was performed in a private hospital on December 23, 1949, under general anæsthesia with "Pentothal", gas (cyclopropane, ether and oxygen) and "Syncurine". During the operation, Dr. S. V. Marshall, who gave the anæsthetic, was puzzled by unaccountable temporary rises in the blood pressure from 150 millimetres of mercury, systolic, and 80 millimetres, diastolic (the pre-operative level) to 220 millimetres of mercury, systolic, and 120 millimetres, diastolic. The patient had a stout, deep abdomen with a small duodenal ulcer in a small, high stomach, which bled at every prick with the needle. After operation, the patient was very well for thirty-six hours. At the end of that time he suddenly became unconscious, and the pulse rate rose to 140 per minute and the axillary temperature to 104·6° F. He had quadriplegia, with small, fixed pupils, and absence of knee jerks and plantar reflexes. He died on the fourth postoperative day. No autopsy was performed. Dr. Diethelm believed that death was due to a basal cerebral hæmorrhage.

Mr. L., aged seventy-four years, underwent operation at Saint Vincent's Hospital on February 28, 1950, for two large gastric ulcers and a duodenal ulcer. He progressed well till March 4, 1950, then suddenly became dyspnœic and cyanosed, with rapid, irregular pulse. Dr. R. J. Taylor diagnosed primary cardiac failure. Dr. H. Windsor thought the symptoms were not of pulmonary origin. The patient died a few hours later. Autopsy revealed the operation area to be normal. The coronary arteries had extremely thick walls and narrow lumen, and coronary insufficiency was thought to be the cause of death.

A study of these deaths at least affords the consolation that none was due to technical failure.

However, there is another side to the question of mortality which is important when one is weighing for a patient the dangers of his disease against the dangers of an operation intended to relieve it. Thompson and Prout (1947) report a mortality rate of 27% in 41 cases at the Los Angeles General Hospital. Bartels and Dulin (1947), from the University Hospitals, Iowa, report a mortality rate of 28% in their first fifty cases, 6% in the next 100, and 1·6% in the last 121 of their 221 cases. In smaller series, unpublished, the mortality rate ranges from 30% to 100%.

The authors mentioned above have performed a considerable service to surgery by illustrating the special nature of gastrectomy. It differs from most operations in that, after removal of the diseased organ, there remains the tricky task of visceral reconstruction, for which the surgeon should have a proper and peculiar sympathy.

Patients with acute hæmatemesis or melæna are admitted primarily to a medical ward. Of 26 patients operated on during acute hæmorrhage, four died. This high mortality would appear to justify Finsterer's teaching that all patients with hæmorrhage from a known chronic ulcer should undergo immediate operation. A difficulty is that many of these patients were not known to have a chronic ulcer, and had even been symptom-free. Perhaps it would be better to make Finsterer's rule even more strict, and to operate immediately on all patients with bleeding severe enough to cause general symptoms, or to necessitate admission to hospital or blood transfusion, irrespective of whether there are other symptoms of chronic ulcer or not; provided always that an expert gastric surgeon is available to operate. One patient had gastrectomy for an acute perforation, and survived.

Early Post-Operative Complications.

Chief among the cases in which early post-operative complications occurred were the following.

Mrs. M., referred by Dr. B. Hall, developed atelectasis, but recovered promptly after bronchoscopy by Dr. H. M. Windsor.

Mr. N., referred by Dr. B. Hall, had severe portal cirrhosis, with portal hypertension. Vascular adhesions attached the liver to a severely gastritic stomach. A second laparotomy was performed for intraperitoneal hæmorrhage of unknown origin, which was found to have stopped spontaneously.

Mrs. O. was referred by Dr. P. L. K. Addison on account of seven severe hæmatemeses from severe chronic gastritis. After operation, her stoma slowly narrowed and required further operation in six weeks.

Mrs. P., referred by Dr. R. Jeremy, had undergone a previous operation, at which a mass in the head of the pancreas had been explored and part removed for biopsy. Later, the mass was found to be due to a large posterior wall ulcer penetrating the pancreas. During operation, an unexpected loop of jejunum, densely adherent to the under surface of the liver and to the anterior surface of the first part of the duodenum, was opened. It was repaired and noted to be of good colour; but it afterwards sloughed en masse, probably on account of mesenteritis and vascular obstruction. Jejunal contents were discharged through the wound. At further operation, two weeks after gastrectomy, the pouting ends of the loop were found in the walls of a large cavity containing slough and jejunal contents. No effort was made to free the two ends, for fear of worse complications, such as duodenal fistula or damage to mesenteric vessels. The jejunum was divided above and below the defect, and an end-to-end anastomosis was performed, so as to exclude the affected loop. The patient is now in good health, except for a fistula which discharges the secretions of the excluded jejunal segment and causes pain when it periodically becomes blocked.

Mr. F., referred by Dr. B. Duffy, had an entero-anastomosis on the fourth post-operative day, for afferent loop obstruction, following gastrectomy and jejunectomy, for a large stomal ulcer with very short afferent loop.

Mrs. R., referred by Dr. Luscombe, suffered an acute coronary infarction thirty-six hours after operation, but recovered. Her huge ulcer is shown in Figure VII.

Mr. S., referred by Dr. F. J. Gray, had a pulmonary embolus with hæmoptysis eight days after operation. On the fourteenth day he had a hæmatemesis, probably due to anticoagulants. His ulcer, almost reaching the pericardium, is shown in Figure VIII.

Apart from Mr. S., and apart from minor blood-staining of the aspirated gastric contents on the first post-operative day, there has been no post-operative hæmatemesis. Nor have there been leaks at suture lines, or wound disruptions.

Acute gastric dilatation was not met with in this series. This complication is sometimes ascribed to perversity of the adrenal glands and depletion of potassium. But this hypothesis appears doubtful, in view of the absence of this complication after such major operations as radical mastectomy. After gastrectomy, it would more probably be due to faults in technique, such as the use of so-called "non-crushing" clamps to prevent spillage, diathermy to transect the stomach, improper placement of stoma and of loops, and crude suturing.

The complications reported here are additional to those reported in 1949.

Intravenous Infusion of Blood and Other Fluids. Rule of Thumb.

If the patient comes to the operating theatre adequately prepared, special methods of resuscitation are usually unnecessary. When unnecessary, they are positively contra-indicated because they introduce complications of their own. Some of these complications, detailed later, are due to intravenous infusion in general. In addition, there are those due to blood in particular. Thus a male patient recently had icterus and pronounced albuminuria during the first week after gastrectomy with transfusion. Other complications are clerical and technical errors, "speed reactions" (for example, pulmonary œdema), pyrogenic reactions, hæmolytic reactions (perhaps due to antibodies undiscoverable by routine matching and cross-matching).

Other patients suffer after some months from virus hepatitis, which may be followed years later by hepatic cirrhosis with a fatal issue. Many of those who readily order "routine" transfusions are debarred, either by youth or by the narrow exigencies of their specialty, from that first-hand knowledge which would impress upon them the reality of later complications. The intravenous infusion of fluids, a major addition to the patient's treatment, should thus be a matter of consultation rather than a matter of light-hearted independence. The clinician should retain preeminence and the controlling influence in the hierarchy, not only because of his broader knowledge, but because it is to the clinician, and to him alone, that the patient has entrusted health and life. Hippocrates, even in his first famous aphorism, stressed this duty and right of the physician.

What, then, is primarily and essentially the indication for transfusion? The answer is: excessive blood loss. In gastrectomy this may occasionally be unavoidable in special circumstances—for example, if a large, high ulcer obscures the left gastric vessels. In other circumstances it should be prevented. Even each of the tiny little bleeders in the skin edge can waste half an ounce of blood. Sweating and tachycardia are not primarily indications for blood transfusion. They are reminders to the anæsthetist to ask "Does the soda-lime need changing?", and to the surgeon to ask, "Am I trying to perform this operation outside instead of inside the abdomen, and, to that end, pulling so hard upon the viscera that the patient would strain and complain severely if local anæsthesia were being used?".

In this series, blood was given during or immediately after the operation in 27 of the 242 interval cases. Even some of these administrations were unnecessary, and blood was given for no other reason than: "I thought it was routine." I was informed by a specialist anæsthetist that it is indeed "routine" in some clinics, not only for gastrectomy, but for radical mastectomy, and even for operations of much less magnitude, such as the repair of hiatus hernia. If blood had been given as a routine in the series here reported, at least 211 patients would have been given unnecessary transfusions, and many litres of blood wasted. No transfusion was given in 214 cases, and 213 of these patients survived. The one patient who died (Mr. L.) did not die through want of blood.

Only two of my patients have received transfusion during or after radical mastectomy, but all have survived.

In the clinics of the great masters of gastric surgery, Eiselsberg, Finsterer, Haberer and others, blood was not given except during acute bleeding from the ulcer. The mortality rate for uncomplicated gastrectomy, and excluding the penetrating ulcers, was from 2% to 4% in the days before the blood bank, before sulphonamides and anti-biotics and modern anæsthesia, and including the malnutrition days of the war years.

If, then, routine transfusion is unnecessary and is given as a rule of thumb to save the effort of deliberation or to forestall the possibility of an evening transfusion, then it should be given up forthwith.

If, however, routine transfusion is necessary, in order to save the lives and achieve a reasonable convalescence for patients after gastrectomy or radical mastectomy or repair of hiatus hernia in any particular clinic, then the surgical technique should be given up.

Even minor injuries, slight and easily supportable blood loss, and even some forms of anæsthesia, lead to a defensive peripheral vasoconstriction, with or without tachycardia. Some anæsthetists tend to give intravenous infusions, rather than a few hours of quiet recumbency, perhaps with elevation of the foot of the bed. If these tendencies progress, transfusions may be prescribed for blood donors.

Another useful method which is nowadays often divorced from reason and debased by routine (possibly, as with blood, because of its ready availability) is the intravenous use of water, glucose and electrolytes. These are now supplied in bottles supplied free of charge by the State. To these have been added synthetic colloids (dextran et cetera) which, when injected into healthy young adults, cause dyspnœa, fullness in the head, precordial discomfort, circulatory disturbance and other symptoms.

Patients after partial gastrectomy should be able to take by mouth sufficient fluids from the first post-operative day. Apart from special circumstances, the necessity for a battery of "Soluvacs" at the bedside suggests that the operative technique has been defective.

Intravenous therapy tends to thrombophlebitis, and this may cause temporary or permanent disability in the leg, even ulceration. Pulmonary embolus, abscesses along the vein, pyæmia and death from a resistant staphylococcus have also been noted.

In the earlier cases in this series, continuous intravenous infusion was given during operation, as a vehicle for anæsthetic agents, and continued after operation; but in recent years this practice has been discontinued. Now, with the anæsthesia in the skilled hands of Dr. S. V. Marshall, or Dr. J. S. Windeyer, or Dr. Brian Pollard, or Dr. R. Killalea, only a minority of patients receive intravenous infusions of any kind after partial gastrectomy.

It belongs to the essence of intelligence to distinguish between things. Attachment to rule-of-thumb is therefore a mark of the less intelligent—or of the lazy.

Late Post-Operative Sequelæ.

Three incisional herniæ developed: a small one at the junction of a transverse incision with its vertical addition; two others during the very short period when I used catgut instead of non-absorbable sutures for the linea alba.

Three patients were admitted to hospital one year or more after gastrectomy, with bilateral pulmonary tuberculosis. One of these, referred from the medical ward, had had gastrectomy for uncontrollable hæmorrhage in chronic gastritis. Another had a chronic gastric ulcer on the basis of a severe erosive chronic gastritis. It is not clear whether these patients had their tuberculosis at the time of gastrectomy. They should be considered in conjunction with the five patients previously mentioned with known tuberculosis at the time of operation.

Type of Operation.

In all cases a modified Billroth II procedure was used.

Gastrectomy-with-exclusion was performed in 18 cases, because the anatomical and pathological conditions at the duodenal end threatened a risky duodenal closure or injury to the bile ducts (Figure IX).

In the British and American literature this operation is wrongly called after Bancroft, a New York surgeon, who wrote about it in 1932. Actually the operation was well established by Finsterer in 1918. On the first page of his first article, Finsterer wrote that "the small stomach stump is closed blindly, after excising as much as possible of the mucosa". He subsequently found that, even if the pyloric mucosa is not removed, stomal ulcer can be prevented by removing enough of the cardiac end.

The tubed-gastrectomy of Schoemaker (The Hague) was used for high gastric ulcer, such as those shown in Figures

VII and VIII, which had almost reached the pericardium after completely penetrating the left lobe of the liver.

I have not used the Billroth I operation (more properly the Gussenbauer-Billroth), because the great masters of gastric surgery, Eiselsberg and Finsterer, considered it to be the less satisfactory operation. Its greatest modern protagonist, von Haberer, gave it up. So also did Hollenbach of Hamburg (1950), after a study of his patients numbering over 2000, and so also did others.

That the conversion of a gastrectomy from Billroth II to Billroth I should sometimes cure post-gastrectomy symptoms in a group of patients, but fail in a larger group, as has been found by Tanner, suggests that in the first group the Billroth II had been improperly performed, and that in the second group the symptoms were not related to operative technique.

End-Results.

The end-results have been very good. There have been no crippling effects of operation in this series, such as have been reported elsewhere. A more detailed consideration of these, and of their prevention, and of substitute methods, will therefore be postponed to a subsequent communication.

Suffice to say here that the substitute methods so far proposed should be abandoned, firstly because they are not necessary, secondly because their after-results will be bad.

Diathermy.

This paper is not primarily concerned with technique; but diathermy has already been mentioned as a probable factor in acute gastric dilatation, and a fuller explanation may be helpful. When used to divide the stomach, it saves much time. But, like many short cuts, it should not be taken. I have not submitted it to a statistical test, for our fundamental knowledge of the natures of things, provided in surgery by the basic sciences, is a more reliable guide than limited statistical series. Under such circumstances, the direct experiment upon human beings is unjustifiable.

When the stomach is divided by diathermy, the resulting edges are coagulated and bloodless, and it is for this reason that diathermy is used. Even before the end of the operation the tissue proximal to the coagulated zone is seen to be swollen, congested and œdematous, with flecks of coagulum floating on it.

Anyone who has had a skin lesion destroyed by diathermy can testify to the excessive reaction and slow healing as compared with an incised wound.

It is amazing that surgeons, who would recoil from the idea of using diathermy on the edge of a pedicled skin-flap, will nevertheless maltreat in this way a sheet of gastric tissue, upon the edge of which a life depends. Attempts are made to meet this objection on the grounds that the stomach is more vascular than the skin. But this justification does not avail, because the edge, after coagulation, is avascular, in skin and stomach. Adjoining tissue, which escapes coagulation, has its circulation seriously damaged, however rich it was beforehand. Vessels do not escape damage just because they are numerous. Beyond this there is a third zone of overheating where the damage is less, but where the capillary permeability is so increased that tissue œdema results. Experiments have shown that this is due to the escape of the large molecules of plasma protein when the temperature reaches 50° C. and increases capillary permeability.

Actually, the lower lip is not far behind the stomach in vascularity, if at all, and A. J. Gardham (1943) advises that diathermy should not be used to excise growths here, as it leads to failure in primary union.

Tissues do not differ essentially in their reaction to crude trauma, and it is therefore of interest to note the remark of Watson-Jones (1952) that not much is gained by the removal of sclerosed bone before bone grafting, if it is merely replaced by burnt bone. Bone one centimetre away may be destroyed by heat from the high-speed cutting tool. But the heat of diathermy is still more penetrating.

It is essential to distinguish between sheets and blocks of tissue. It may be justifiable to use diathermy for hæmostasis in brain, thyroid or breast. In a three-dimensional block the current is widely dispersed, and the zones of damage are minimal; but the current has not such scope for dispersal in a sheet of tissue, and the damage is more extensive. The zones of damage are as follows: (a) the zone of division with complete disappearance of tissue; (b) the zone of hæmostasis, coagulation and complete devitalization with subsequent sloughing or absorption of dead tissue; (c) the zone of severe burning with delayed healing; (d) the 50° C. zone with escape of plasma protein and œdema.

The advocates of diathermy claim that a ligature devascularizes the tissue in its grasp. But the tissue is only devascularized, and it remains as a tiny living graft, embedded in tissue with an undamaged circulation. It is not devitalized, to remain as a coagulum, implanted in tissue with a damaged circulation.

The superiority of the ligature over the cautery was established centuries ago by Paré. Today his words have added significance, because diathermy heat is generated within the tissues themselves, and spreads far more deeply.

> It is a thing which savours not of a Christian, to fall to burning at the first dash without staying for any more gentle remedies.

> Let us come now to Reason. Now so it is, that one cannot apply hot irons but with extreame and vehement paine in a sensible part, void of a gangrene, which would be cause of a Convulsion, Feaver, yea oft-times of Death. Moreover, it would bee a long while afterwards before the poore patients were cured, because that by the action of the fire there is made an eschar, which proceeds from the subject flesh, which being fallen (cast-off), nature must regenerate a new flesh instead of that which hath been burned. . . . Moreover, there is yet another accident. It happeneth that often times the crust being fallen off, the blood issueth out as much as it did before. . . . For the generation of a crust proceeds from the parts subject (subjacent) and which are situate round about it, being also burned, as I may say; wherefore by how much the part is burnt, by so much it looseth the naturall heate.

It is clear that Paré appreciated the devitalization of tissue, which spreads through the heat, beyond that part completely destroyed. He then lays down the rule that the cautery is to be used for the destruction of diseased tissue, not of healthy tissue, and this should still hold good today.

> In fresh bleeding wounds there is neither gangreene nor putrefaction. Therefore, the cauteries ought not to be there applied. And when the Ancients commanded to apply hot irons to the mouthes of the vessells, it hath not been onely to stay the flux of blood, but cheefely to correct the malignitie or gangreenous putrefaction which might spoile the neighbouring parts.

Summary.

1. Partial gastrectomy was performed for ulcer and gastritis in 268 patients. During the same period, gastroenterostomy was performed in 16 cases, and vagotomy in eight others.

2. In 242 interval cases, four patients died (1·6%). In none of these cases was death due to a fault in technique. In the last 161 consecutive cases, no patients have died. The interval cases include eight patients who had undergone previous gastro-enterostomy or inadequate gastrectomy.

3. Of 26 patients in acute hæmorrhage, four died (15%). This high mortality rate could possibly be avoided by a more strict application, and a slight modification, of Finsterer's rule.

4. Two out of three patients needing gastrectomy were males. Among them, the mortality was higher than that among their "weaker" sisters. All deaths in the interval cases, and all but one in the bleeding cases, were of males.

5. The ages ranged from twenty-one to seventy-seven years, the greatest number of patients being in the fifth and sixth decades.

6. In one interval case, early in the series, the patient developed a stomal ulcer on account of a technique which would not now be used.

7. Other complications have been few and minor. The end results have been good. It would therefore appear that substitute methods are unnecessary. Those so far proposed are inadvisable.

8. The intravenous administration of fluids, including blood, was needed in only a small minority of cases. If blood and other fluids are found to be really necessary as a routine, the surgical technique should be revised. If blood had been given as a routine in this series it would have entailed a grave wastage.

9. Division of the stomach by diathermy inflicts unnecessary damage upon the gastric wall, is dangerous, and is not in the best tradition.

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Legends to Illustrations.

FIGURE I.—Large gastric and duodenal ulcers, four years after gastro-enterostomy.

FIGURE II.—Stomal ulcer and obstruction following localized excision of lesser curvature ulcer and gastro-enterostomy.

FIGURE V.—Mrs. E.: post-operative skiagram.

FIGURE VI.—Mrs. E.: stomal ulcer.

FIGURE VII.—Huge gastric ulcer, removed by Schoemaker modification of Billroth II procedure.

FIGURE VIII.—Lesser curvature ulcer, almost to pericardium (Mr. S.).

FIGURE IX.—Duodenal ulcer penetrating bile ducts, treated by Finsterer's gastrectomy with exclusion.

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IMMEDIATE GASTRECTOMY FOR PERFORATED PEPTIC ULCER.

By DANIEL LANE, F.R.C.S. (Edinburgh),
F.R.C.S. (England), F.R.A.C.S.,
Brisbane.

AT the present time surgical literature is being flooded with a spate of articles on the topic of gastrectomy and the post-gastrectomy syndrome. Most of us are convinced (except perhaps some who have undergone the operation) that partial gastrectomy is a successful procedure in the majority of cases. The chronic ulcer which is causing severe pain is worthy of early resection; the bleeding ulcer and the stenosing ulcer are well treated by gastrectomy under certain conditions. What, then, of the place of gastrectomy in the treatment of the perforated ulcer?

The opposing forces of conservative and radical surgery meet in furious onslaught over this problem. Many continental authorities have long favoured immediate resection; American opinion is becoming more disposed towards this method of treatment; but the British school has largely opposed the idea, preferring to remain tradition-

ally conservative. There must be a *via media*; let us briefly analyse the problem. I will presume that most of us favour some form of operative treatment of perforated peptic ulcer.

Introduction of Partial Gastrectomy for Perforated Peptic Ulcer.

Partial gastrectomy for perforated peptic ulcer was probably first performed by an Englishman, Keetley, in 1902. However, most of our inquiries on this topic must be directed to certain continental sources, where the operation has proved remarkably successful. The late Sergei Yudin had a mortality of 9% in 937 cases, which is remarkable when we consider that many of his patients were operated upon before the advent of many modern pre-operative and post-operative aids now accepted as routine.

To the Dutch school we must turn for many authoritative recent writings on the subject. Eerland, Nuboer, and recently Quast (1955) have added strong support to this radical form of treatment.

Objections to Gastrectomy.

For various reasons one may oppose the use of immediate gastrectomy for perforated peptic ulcer. (i) The old teaching of "treat an emergency as an emergency" is usually sound. We are asked to deal primarily with peritonitis, not with an ulcer. Let us close the perforation and treat the ulcer later. This argument is certainly correct in many cases, especially in relation to young people. However, there can be no objection to partial gastrectomy on the grounds of higher mortality. The results of Yudin, and especially those of Nuboer and Quast, show that the operation is a safe one and that experienced surgeons can achieve a very low mortality rate. Maybe we have been hindered in our therapeutic progress by adhering to conservative simple closure as a routine procedure. (ii) In 30% to 40% of patients, when perforation has undergone simple closure the ulcer will remain healed. Partial gastrectomy would be unnecessary if employed in such cases. Upon this we are all agreed. However, let us not forget that almost 60% of patients who have sustained a perforation will suffer further symptoms at a later date. In a small proportion recurrent perforation will follow. If conditions permit, why not then effect a cure at the first operation? (iii) In a fairly high proportion of cases the patient with a perforated peptic ulcer gives a history of ulcer symptoms for only a short time before the perforation. If the patient is young and his perforation small, most of us would be loath to perform gastrectomy as an immediate measure. However, certain continental authorities would not raise any great objection to such a radical approach. Nuboer argues that most ulcers that perforate are, in fact, chronic, and not acute as we are often led to believe. Therefore he claims that partial gastrectomy will rid the patient of a chronic ulcer, and most of us will admit that such an ulcer is always best treated by partial gastrectomy. This argument depends largely upon the histological interpretation of perforated ulcers. (iv) Most conservative surgeons consider that partial gastrectomy is unnecessarily severe for perforated ulcer. "Why take undue risks?" they would argue. However, to most experienced general surgeons partial gastrectomy presents no extraordinary difficulty today. An operating time of two hours and less can be readily achieved. Experience of large series of cases has shown that partial gastrectomy in these cases is tolerated extremely well—sometimes even better than closure of the perforation alone.

Those surgeons who have performed the operation as an immediate measure in the presence of perforation will agree that frequently the operation is rendered easier by virtue of the presence of œdema of the tissues. To quote from Aird:

> Even a perforated simple peptic ulcer of the stomach may, if it is extensive and so surrounded by œdema that stitches cut out, be treated more simply and gently by gastrectomy than by simple closure.

The Indications for Immediate Partial Gastrectomy in Perforated Ulcer.

Provided that the general condition of the patient is good and that the local abdominal condition allows of resection, one is justified in proceeding with partial gastrectomy in certain instances, namely: (i) in almost all perforated gastric lesions; (ii) when the perforation is large and simple closure would seem unsatisfactory; (iii) in the presence of concomitant hæmatemesis and stenosis; (iv) when a history of previous perforation is given; (v) if there is a long history of dyspepsia, especially in middle-aged and older subjects.

It must be presumed that the surgeon feels quite competent to carry out an efficient and speedy gastrectomy, say in two hours or less, and that hospital facilities are completely adequate.

Such factors as old age and perforation time beyond a certain arbitrary period are important only in so far as they influence the general condition of the patient, and are not necessarily deterrents to radical operation.

The Success of Primary Resection.

According to one's beliefs, one can advance many arguments and show many figures to support the various methods of treatment of perforated peptic ulcer. As Edwards puts it: "Results are as nearly equal as to make argument profitless as to which is the best." If we can achieve earlier diagnosis and admission to hospital, a skilful surgeon can produce excellent results by the method of primary resection. Perhaps we have not given this method a reasonable trial. If surgeons like Nuboer and Quast can achieve an overall mortality rate of less than 4%, then the operation is a safe one.

In a recent series of 344 cases Quast has reported an overall mortality rate of 3.2% in 150 cases of primary resection. He illustrates the considerable improvement in results which has occurred since the advent of the "antibiotic era" (after March, 1947). Before March, 1947, the mortality rate was 6.1%, and since March, 1947, in 90 consecutive primary resections the mortality rate was only 1.1%.

Reports of Cases.

The following two brief case histories illustrate the problem under discussion.

CASE I.—In December, 1953, a man, aged seventy years, a pensioner, was admitted to hospital complaining of severe epigastric pain which had commenced suddenly two hours previously. In 1952 he had suffered from hæmatemesis, but X-ray examination with a barium meal failed to reveal any ulcer, although one was strongly suspected. In October, 1953, a further barium meal X-ray examination failed to reveal any abnormality. Physical examination revealed the patient to be a surprisingly fit-looking old man who was in great distress. The abdomen was absolutely rigid, and there was loss of liver dullness. A diagnosis of perforated peptic ulcer was made. Immediate laparotomy revealed a small perforation in the anterior wall of a very large prepyloric ulcer which was invading the pancreas. In view of the general fitness of the subject and of the presence of such a large prepyloric ulcer (which had apparently caused the previous hæmatemesis), immediate partial gastrectomy was decided upon. An ante-colic Pólya type gastrectomy was performed. The patient made a completely uneventful recovery. The pathological examination revealed a deep chronic ulcer measuring 3.0 by 2.0 by 1.5 centimetres. There was a perforation 10 by 5.0 millimetres in area in its anterior wall, and there were several large thrombosed arteries in the base. Examination of the sections revealed a chronic active gastric ulcer without evidence of malignancy.

In this case the perforated ulcer had caused bleeding a short while previously, and owing to its size and prepyloric position malignant change was considered possible. In view of this and of the general fitness of the subject, a radical operation seemed well justified. I am quite certain that the operation was far easier than it would have been if the gastrectomy had been delayed until some months later (presuming that the patient had recovered by then). His post-operative recovery was so uneventful that one formed the impression that simple closure of the perfora-

tion could not have been followed by a more satisfactory convalescence.

CASE II.—A housewife, aged fifty-three years, was admitted to hospital in May, 1955, with a history of sudden onset of abdominal pain six hours previously. She admitted to having suffered from a duodenal ulcer for many years. She had lost at least two stone in weight during the preceding eighteen months. During the past three to four months she had suffered from frequent bouts of vomiting. Examination revealed her to be a thin, dehydrated woman in great pain, with board-like abdominal rigidity. There was no evidence of undue shock. A provisional diagnosis of perforated peptic ulcer associated with pyloric stenosis was made.

Immediate laparotomy revealed gross peritoneal soiling due to a large perforation of an anterior wall prepyloric ulcer. In addition, the stomach was much hypertrophied from the presence of established pyloric stenosis. Owing to the presence of stenosis distal to the perforation, simple closure of the perforation seemed unsafe, and as the patient's general condition was reasonably satisfactory, immediate partial gastrectomy was considered to be the ideal solution to the problem. An ante-colic Pólya type gastrectomy was performed without undue difficulty. The patient made a very satisfactory recovery. On the sixth post-operative day it was thought that a right-sided posterior subphrenic abscess was developing, but her symptoms and signs rapidly subsided, and no further treatment was required.

The pathological examination of the perforated prepyloric ulcer revealed intense acute and subacute inflammation, but no evidence of malignancy. There was well-established pyloric stenosis, almost certainly due to a healed duodenal ulcer.

Here again were circumstances which immediately suggested partial gastrectomy as the ideal answer to the problem. The patient's satisfactory recovery fully justified this radical step. She is now able to resume normal activities without thought of further operation.

Comment.

The best method of treatment of perforated peptic ulcer is still a subject for controversy. In Australia most surgeons would undoubtedly favour simple suture with or without an omental graft. For the occasional operator and in isolated areas with restricted facilities this is certainly the wisest course to follow.

However, in the larger and better equipped centres the experienced surgeon can perform partial gastrectomy as an immediate measure with excellent results, quite comparable with those obtained by simple suture. One cannot agree with the ultraradical approach recommended by many continental authorities. After all, 30% of all perforated ulcers do not cause further symptoms, and immediate gastrectomy would be quite unnecessary in such cases.

Although as yet no long-term follow-up figures are available, it becomes increasingly apparent that we are justified in performing immediate resection more often than hitherto.

The arguments in favour of resection have been summarized by Quast, and, with certain modifications, are as follows:

1. The results are good in selected cases.

2. A possible second operation is avoided.

3. The operative risk has been reduced considerably nowadays. Any capable general surgeon with adequate facilities should be able to achieve a small mortality rate.

4. The diagnosis is not always certain in the beginning. A certain proportion of perforated gastric ulcers are carcinomatous and demand immediate resection.

5. Many complications are eliminated. These include continued leakage, bleeding and stenosis.

6. In the majority of patients aged over fifty years a chronic peptic ulcer becomes a major hazard to life. If immediate removal of this hazard can be performed successfully at the time of perforation, it would seem well justified.

Summary.

A brief review of the arguments for and against primary resection for perforated peptic ulcer has been presented.

We must preserve a balanced outlook to the problem. Our personal bias for one particular method of treatment deserves healthy analysis. Perhaps our results can be improved by adopting the more radical operation in many cases.

Two case histories have been included to illustrate circumstances which fully justify immediate resection as a method of treatment of perforated peptic ulcer.

Addendum.

Cooley and his associates (1955), presenting some recent American views, have analysed 199 cases of acute gastroduodenal perforation and report that for 112 cases treated by gastrectomy the total mortality was 4·5%; for 70 patients treated by simple closure, the mortality rate was 8·6%. These figures lend further support to the practice of gastrectomy in the treatment of a large proportion of gastroduodenal perforations.

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THE "COLON CUT–OFF" SIGN IN ACUTE PANCREATITIS.

By C. W. R. PRICE,
Perth.

IN a paper which was published a few years ago, but which, to my regret, I cannot now trace, there was recorded the description of an X-ray sign which is often diagnostic of acute pancreatitis. In the early stages of an attack of pancreatitis, often within a few hours of the onset, the plain abdominal X-ray film shows an isolated gaseous distension of the ascending colon and hepatic flexure, with sharp delimitation of the gas shadow just to the left of the flexure. "Colon cut-off" has seemed an appropriate name for the sign.

Shortly after this paper was published, a laparotomy was performed on a patient, Mr. A, whose radiograph appears in Figure I, and it was observed that there was a cellulitis-like process spreading from the œdematous head of the pancreas between the two layers of the transverse mesocolon to reach the mesocolic border of the colon just to the left of the hepatic flexure. Clearly the inflammatory process had determined an obstruction at this point, sufficient, at least temporarily, to hold up the onward passage of gas.

Figure II (after "Gray's Anatomy") shows the attachments of the transverse mesocolon, indicating the anatomical basis of the observation. Figure III is an impression of the appearances at operation.

The X-ray films of two patients, Mr. B (Figure IV) and Mr. C (Figure V), also show the "cut-off" sign. In the case of Mr. B the diagnosis and the mechanism of the sign were demonstrated at operation. In the case of Mr. C the diagnosis was not confirmed by exploration, but by a raised serum diastase level, by the subsidence of symptoms under conservative treatment, and by the recurrence of attacks under the provocation of alcoholic excess.

A barium meal X-ray examination was made on Mr. C in the early stages of one of his attacks; this demonstrated the duodenal ileus well known to occur in some cases of pancreatitis. Reference to Figure II suggests that this duodenal ileus may well be determined by a similar extra-
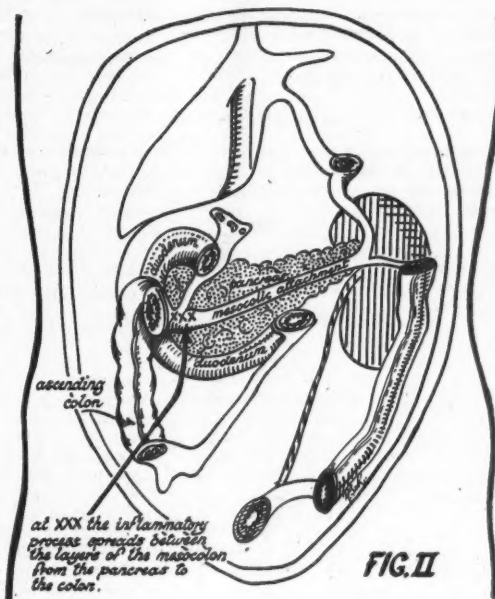
FIG. II

peritoneal spread of inflammation to the duodenum at about the junction of its second and third parts where it is related to the root of the mesocolon.

The frequent finding of localized ileus of the small intestine may have a similar basis in the spread of inflam-
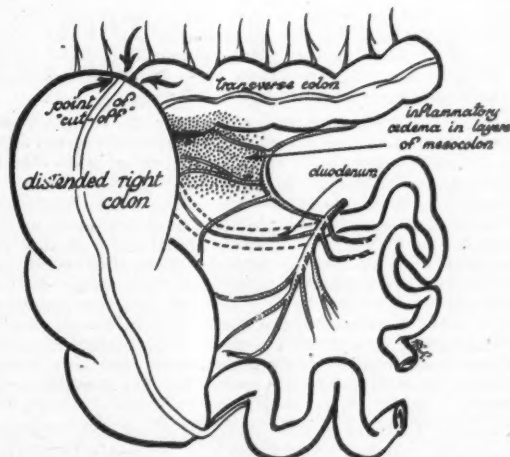
FIG. III

matory œdema between the leaves of the small intestine mesentery, but I have not confirmed this at operation.

My radiological colleague at the Fremantle Hospital, Dr. Charles Stuart, M.C., has traced many other examples of the "cut-off" sign and is preparing a paper in greater detail than the present communication.

As has already been noted, no claim is made to priority in the description of this sign, and I owe my apologies to the authors of the original paper. However, I have not found any previous account of the underlying pathological anatomy.

Summary.

1. A radiological sign sometimes diagnostic of acute pancreatitis is redescribed.

2. The anatomical basis of the sign as demonstrated at laparotomy is described.

Acknowledgement.

I have pleasure in thanking Mr. R. B. van Raalte, of the Royal Perth Hospital, who is responsible for the sketches as well as for the prints of the X-ray films.

PRE–OPERATIVE AND POST–OPERATIVE MANAGE-MENT OF THE NEWBORN: INTRODUCTION AND SURGICAL CONSIDERATIONS.[1]

By J. STEIGRAD,
Sydney.

WHEN invited to take part in the proceedings tonight on the subject of the pre-operative and post-operative management of the newborn, I accepted with alacrity, because, as a representative of the surgical staff of the Royal Alexandra Hospital for Children, I am anxious that the surgery of the newborn be accorded the widest publicity within the profession. More particularly am I happy to speak tonight because assisting in this round table conference are Dr. John Beveridge, the chief resident medical officer of the Royal Alexandra Hospital for Children, and Dr. Andrew D. Morgan, the senior visiting anæsthetist of the hospital.

Tonight is again a very good oppportunity to indicate generally how great a debt we, the surgeons, owe to the senior house staff and to the specialist anæsthetists of the hospital: to the latter, for the high standard of anæsthesia which we now have come to regard as an integral and routine part of our work in the surgery of the newborn; and to the former for the painstaking care which they give to their task of intravenous resuscitation of these tiny patients and of maintaining them on an even keel in the turbulent sea of electrolyte balance.

The results of surgery of the newborn are in this State showing a gradual improvement as regards the survival rate, although they still fall far short of the results obtained by surgeons in the United States of America. It is considered that this improvement is closely related to four factors. These are: (i) A better understanding of the underlying pathology of neonatal conditions, a finer technique and a more daring and purposeful approach by surgeons. (ii) Recognition of serious neonatal conditions at an earlier age by family doctors and pædiatricians. (iii) A more intimate knowledge of the fluid and electrolyte balance in infants and a more expert application of this knowledge by the senior house staff. (iv) Good anæsthesia by anæsthetists experienced in the anæsthesia of infants.

I propose to make a few observations on the first and second of the above factors in their relationship to surgery in the newborn. The third and fourth factors will be in the hands of two colleagues who are as experienced in these fields as any in this State. Dr. Beveridge will discuss the care of the newborn infant from the diagnosis or the suspected diagnosis in the obstetric hospital to the time when the surgeon has decided to operate and the

infant is deemed to be fit for surgery. Dr. Morgan will stress the immediate pre-operative care, and from his own experience will describe the anæsthetic agents and techniques, resuscitation in the operating theatre, and post-operative care until the infant is considered fit to return to its cot. Dr. Beveridge will then detail the post-operative requirements as to gastric suction, intravenous feeding, oxygen inhalation *et cetera*.

In this method of presentation, I hope that you will appreciate the very crux of the problem, which is close association and cooperation to the extent of full teamwork by pædiatrician, anæsthetist and surgeon. This teamwork implies that the members of the team together will examine the patient as often as is necessary and confer together. This is because, despite all instructions, memoranda and tables, an assessment of the condition and of the requirements of the patient depends ultimately upon that difficult to define clinical attribute—experience.

Surgeons in this country are indebted to men overseas like Gross, Svenson, Ravitch, Denis-Browne and many others for the publication of the results of their experience in the surgery of the newborn. The results that are obtained in the United States of America are a challenge which we are continually striving to accept and match. However, if we are to reach this goal then the second factor I mentioned, that of earlier recognition, must be stressed. Medical practitioners should suspect hidden disease in the newborn and should realize that, frequently, early surgery offers a real hope of cure. All too frequently is the surgeon faced with the care of an infant who is already well-nigh overwhelmed by the malignant effects of dehydration, inanition and toxæmia.

In the newborn, conditions which require surgery fall into several groups. These are (i) congenital abnormalities of the alimentary tract from œsophagus to anus, (ii) congenital abnormalities which threaten respiration, such as diaphragmatic hernia, bronchial hypoplasia and micrognathia, (iii) miscellaneous conditions, such as malignant growths, exomphalos, ruptured or threatened rupture of a myelomeningocele, (iv) infective conditions, hæmatogenous osteomyelitis, abscesses and empyema.

A newborn infant who is not very premature and is in a good general state is an excellent subject for surgery. He takes good anæsthesia well, is not prone to post-operative infection, and raises no complaints so long as his hunger is appeased and kept so. His post-operative convalescence is generally pleasant and uneventful. However, if the general state is not good and if the operative procedure proposed is of some magnitude, operative shock is a factor which must not be forgotten. The use of a venous cut-down and the running of blood into the vein during an operation should always be considered. The procedure of short duration or of a non-traumatizing nature, such as a Ramstedt pyloro-myotomy or an inguinal herniotomy, naturally generally requires no operative transfusion. However, if the procedure is likely to be lengthy, if the whole detail of the technique cannot be known at the outset, or if there may be blood loss, then blood should be running into the vein during the operation. An infant at operation may, and does, tend to lose more blood than the surgeon may realize. A newborn infant cannot withstand the loss of blood and the surgeon may well be astonished at the quantity of blood that an infant may accept during an operation.

Because of the great difficulty in assessing the degree of post-operative shock in a newborn infant in the theatre, it is a very wise precaution to attempt to prevent its onset by the use of a blood transfusion. If an infant has evidence of dehydration, the operation should wait for an hour, two hours, or even twelve hours, until adequate rehydration is achieved. This is the wisest course to follow unless the operation is of immediate urgency, such as one for the relief of respiratory distress due to a diaphragmatic hernia.

I will say nothing about general anæsthesia except to make the remark that one realizes how desirable good

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anæsthesia is in these babies only when one does not have it. As regards local anæsthesia, much surgery is possible with local anæsthesia, but I am one who believes that local anæsthesia alone never replaces that state of blessed tranquillity for patient and surgeon alike which comes from general anæsthesia administered by an expert.

From the surgeon's point of view in the matter of pre-operative and post-operative care of the newborn two salient features stand out. One is that good results will come only with the closest consultation between surgeon, pædiatrician and anæsthetist, and the other is concerned with operative and post-operative shock. These are so difficult to assess that all measures directed towards their prevention are most important.

PRE–OPERATIVE AND POST–OPERATIVE CARE OF THE NEWBORN.[1]

By A. DISTIN MORGAN,
Sydney.

TEAMWORK of the highest order with a complete understanding between all concerned is probably the greatest contribution which can be made to the recovery of infants requiring surgical treatment. There is no stage at which this is more important than before the operation. Once the diagnosis has been made and the anæsthetist has been summoned, there should be full discussion concerning the nature and extent of the surgical interference, the anæsthetic to be employed, and the general condition of the patient. At this stage it may be well worth while to delay the operation in order to improve the condition of the little patient. If dehydration, blood loss or shock is evident, time should be taken to correct any or all of them, so that the patient is presented for operation in the best possible condition. Should respiratory insufficiency be present, it should be relieved, and, according to the degree and cause, the necessary relief may entail anything from the use of oxygen in a tent or catheter to intubation of the larynx with positive pressure respiration. It frequently happens that these infants have been undergoing some form of investigation involving the use of opaque materials and X rays, with consequent exposure to cold. Failure to delay the start of the operation before the barium is completely removed and the child is warmed up may well prove the deciding factor in the recovery or in death from irreversible shock. The child's stomach should be emptied of food or barium, and shock and heat loss should be corrected before operation is commenced.

Premedication.

Sedation is generally considered to be unnecessary unless the child is causing itself damage by failure to rest, in which case some sedation may be desirable. Atropine is considered both to protect the child from the shock which may be caused by the handling of the gut and to lessen the dangers of the reflex vagal effects. Atropine also controls secretions, which may be especially troublesome at this age. A dose of $1/600$ grain to $1/450$ grain should be given forty-five minutes before operation.

Nature of Anæsthetic.
Drugs.

Local anæsthesia alone is seldom satisfactory, but as a prelude to general anæsthesia it is valuable for the early stages of an operation when the general condition of the patient is poor. Infants with abnormalities such as tension emphysema, diaphragmatic or hiatus hernia, or other conditions which give rise to respiratory embarrassment, may be handled in this way. A local anæsthetic is administered and intubation is effected. Little additional anæsthetic

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[1] Read at a meeting of the Section of Pædiatrics of the New South Wales Branch of the British Medical Association on May 3, 1955.

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may be needed, but if so it is now safe, because the lung can be inflated when the thorax is open. If inflation was to be attempted in a patient with tension emphysema before the chest was opened, the already dangerous respiratory insufficiency would be made worse, because the emphysematous portions would continue to expand at the expense of the rest of the lung. Other anæsthetic agents can be used according to choice and circumstances. If the diathermy is to be used during the operation, explosive gases must be avoided. If the diathermy is not to be used the choice is wide open. Gross and many others advocate ether, and we have found this to be quite satisfactory. Cyclopropane and nitrous oxide have proved to be satisfactory. Chloroform and ethyl chloride are considered to be too toxic and too sudden in their effect and are therefore not used for patients of this age group. Intravenous and rectal thiopentone have been tried but give uncertain results and should generally be discarded. Relaxants in appropriate dosage have proved to be a most satisfactory addition to anæsthesia. Many surgical procedures have been undertaken in neonates using relaxants without any anæsthetic, and employing intubation with respiratory control. Whatever drug is used, the dose should be minimal, the airway must be completely free and the oxygen should be maximal.

Technique.

An endotracheal technique must be employed if the thorax is to be opened. It is also desirable for most other operations in this age group. If necessary, intubation can be performed without anæsthesia.

The method of managing the endotracheal anæsthetic technique is important, for both dead space and resistance must be reduced to the absolute minimum. The circle of an ordinary gas machine is useless because it does not provide sufficient precision of control. If a "to-and-fro" method of anæsthesia is desired, a small cannister of 100 cubic centimetres capacity or less is necessary, together with a very small bag. A simple method which fulfils most requirements is provided by using an "Ayre's T piece". Controlled respiration with this is carried out by simply occluding the open end and opening it rhythmically.

Shock and Resuscitation.

The temperature of the patient may be elevated or depressed. If the temperature is elevated it is well to recall that each one degree rise increases the oxygen requirements by 7%. Failure to give oxygen to an infant with a raised temperature may be the means of provoking convulsions. Heat loss is also of great importance. In such tiny patients it is hard to expose a sufficient area for operation without exposing most of the child's body, and heat loss can easily occur. Under these conditions in a cold theatre the loss of heat can be considerable.

Blood pressure estimations in infants at operation may be unreliable and difficult to obtain, and the degree of damage caused by blood loss and handling may be hard to detect. Indeed, the clinical recognition and evaluation of shock may be impossible; additionally an infant's protective mechanism keeps the child looking well until the cerebral centres become depressed by anoxia. By the time shock has become obvious it may be irreversible. Experience has shown that these patients, with very few exceptions, all need transfusion, and the amount is probably greater than previously has been thought necessary. Two hundred to 250 cubic centimetres of fluid by the intravenous route is not excessive even if blood loss is minimal. It is common practice for a fluid such as glucose solution, with or without saline in various strengths, to be dripping into a vein at the commencement of operation. Blood is withheld until later, when it may be considered to be necessary. It is well to recall that the drip tubing holds about 30 cubic centimetres, so that at least this amount of fluid has to be run through before blood reaches the patient. This causes loss of valuable time, and it may be the means of giving too rapidly a volume of fluid less useful for shock therapy than blood itself, and also it

delays considerably the subsequent arrival of the blood in the patient's veins. Therefore blood must be actually entering the patient's veins when the operation is commenced. The rate at which the fluid drips may be as slow as required. When blood is needed the drip rate has only to be increased for additional blood to be immediately available to the patient. It is important that blood loss should be estimated as nearly as possible as it occurs, and adequate replacement should be made immediately.

The recovery rate for patients undergoing operation in this age group, estimated from our own records and from the records published by others, is only about 30%. Of those that die after operation, 50% perish in the first twenty-four hours. This suggests that our methods of resuscitation have been inadequate and need to be improved. Many who die in the post-operative period appear to leave the operating theatre in a satisfactory condition, but soon afterwards they develop obvious signs of shock and need resuscitation. Besides the more frequent and liberal use of blood during operation already advocated, the avoidance of changes of posture and of excessive handling is considered to be important in the prevention of the post-operative onset of shock. All possible measures should be employed to avoid movement, handling, heat loss, and deficient oxygenation. The replacing of garments after operation necessitates much movement and could well be omitted for some hours. The patient's bed should be brought to the theatre and once back in it the infant should be given oxygen from a cylinder attached to the bed. There is little doubt that improvement in the recovery rate would be greatly assisted if infants after operation could be nursed in a special shock ward by highly skilled and experienced staff. By no other means can the experience be gained and the nicety of judgement be developed to such extent that the earliest departure from the norm is recognized and appropriate treatment is instituted immediately. The earlier recognition and thorough treatment of shock can improve the outlook in these cases.

PRE–OPERATIVE AND POST–OPERATIVE CARE OF THE NEWBORN.[1]

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SOME of the difficulties which confront those who assist in the management of neonates requiring major operation are the same as those involved when operation of comparable magnitude is performed on older patients. Certain of these problems are, however, peculiar to the newborn period.

General Considerations.

A congenital malformation is the most frequent indication for surgery in this age group. Not uncommonly where one congenital abnormality is present, other abnormalities are also found. Congenital heart disease in association with mid-line deformities, such as intestinal atresia or tracheo-œsophageal fistula, and fibro-cystic disease of the pancreas (which regularly accompanies meconium ileus), are examples of associated abnormalities which are often sufficiently major to cause the death of the infant in spite of successful surgical correction of the other condition. In infants in whom duodenal obstruction is associated with mongolism, one wonders if one should strive too officiously to keep the child alive.

Certain aspects of physiology in the newborn are of great interest. Adrenal function appears to be depressed. Circulating corticoids, presumably derived from the mother,

on the first day of life are at adult levels. The level falls on the second day and is greatly depressed, until the end of the first week (Kelly, 1955). According to Klein (1954), no corticoids are detectable during this period. At the end of the first week the reading begins to rise and it reaches a normal childhood level at the end of the second week of life.

The kidney function of the newborn child is immature. Urinary output in the first few days of life is small, and the concentrating power of the kidney at this time is rather less than half that of the adult.

Control of body temperature is also immature, and the newborn infant can readily be chilled or overheated by variations in external temperature. Chilling is the more frequent problem in clinical practice. Despite present thought on hypothermia in anæsthesia, it has been my experience that chilling is undesirable in this age group and may be fatal.

At least two aspects of blood coagulation are disturbed in the first few days of life: prothrombin and factor VII levels are depressed. Recent evidence suggests that factor V (Israels et alii, 1955) may also be at a level lower than that of older children. Although parenteral administration of vitamin K may raise the prothrombin level, transfusion of plasma or blood is indicated, because of coagulation defect, when any surgery is undertaken at this time.

The vital capacity can be grossly impaired by diaphragmatic hernia, which is frequently a surgical emergency, by tension emphysema, and by abdominal distension due to intestinal obstruction. Abdominal distension is also important because it may progress on to rupture of the gut, because it represents a mechanical embarrassment to to the surgeon, and because distended gut does not readily resume its function even when obstruction has been relieved by surgical measures.

Infection is a serious problem in the inhalation pneumonia which is a constant complication of tracheo-œsophageal fistula, unless repair is undertaken very early. Meconium peritonitis, a complication of rupture of the gut due to intestinal obstruction, is even more serious.

Fluid and electrolyte balance and the state of the circulation are of great significance. The normal neonate may lose from 6% to 10% of its body weight during the first week of life. Allowance is made for this when the clinical state of hydration is being assessed. Losses of fluid and of electrolytes from the kidney are small, and normal requirements of fluid and electrolytes are also small; from the technical point of view the volumes involved are minute. The blood volume of the normal neonate is of the order of 300 millilitres and the total extracellular fluid volume is only a little more than a litre. Because of these factors, infinite care and skilled judgement are required in assessing the amounts of fluids, containing electrolytes, to be given. However, the apparently minute blood loss which occurs at operation is best replaced in full as it occurs.

Nutrition can be a serious problem when oral feeding cannot be undertaken for technical reasons until the baby is two weeks of age or even older. It is an even greater problem when one is confronted with the demands on body protein metabolism made by infection, or when one is faced with the malabsorption syndrome of fibrocystic disease of the pancreas.

The premature infant does not present with any different problems, but it is readily apparent that all the difficulties of physiology and of technique are rather greater than in the full-term infant.

Pre-Operative Period.

In the interval between the time when some abnormality of the newborn baby is suspected to the moment when the patient is taken into the operating theatre, the aims of the physician are to achieve an accurate diagnosis and to have the child in the best possible condition for operation. The physician keeps in mind the hazards of air travel for

babies whose vital capacity is diminished, and for those whose intestines are so distended that increase in altitude may cause the gut to rupture. In such cases it may be advisable for the physician, surgeon and anæsthetist to go to the baby rather than have it brought to hospital. The physician supervises the stage of investigation, he protects the baby with tracheo-œsophageal fistula from unnecessary laryngoscopy, and he avoids unnecessary exposure and chilling of the infant if fluoroscopic screening of the chest and abdomen, or barium studies, are essential in order to reach a diagnosis.

Except for patients with a congenital diaphragmatic hernia, pre-operative preparation may extend over several hours. Urgent life-saving measures are sometimes necessary; most often such urgent measures are directed towards the respiratory system. Oxygen is indicated for babies in whom there exists either tension lobar emphysema or diaphragmatic hernia. In this latter condition I have found it necessary to pass an endotracheal tube and perform positive pressure respiration until operation could be undertaken. The baby with tracheo-œsophageal fistula requires posturing in the head-down position and frequent oropharyngeal suction; for an occasional patient, urgent laryngoscopy and bronchoscopy may be needed.

When abdominal distension is present, gastric intubation is performed as early as possible. The technique of gastric intubation is too often taken for granted; in the newborn (and in children at all ages) Rule's tube is unsatisfactory, and a large bore catheter, in which several side-holes have been cut, is preferably used for the intubation. The tube is frequently too large to pass through the nose, and is passed instead through the mouth. The distance from mouth to stomach is measured and marked on the tube, to ensure that it is passed to the optimum distance. Continuous suction of the gastric contents is not as good as frequent intermittent suction by means of a syringe.

A surprising number of these newborn babies who require surgical treatment become chilled before reaching the hospital. Their temperature cannot be recorded on a clinical thermometer and the heart rate is 100 per minute or less. Such a patient can be warmed slowly over six hours or more, if time permits, before operation is undertaken. This warming is achieved by hot-water bottles, used as in the bassinette of a premature baby.

Occasionally a patient is shocked by blood loss prior to operation. Such patients are the only ones to whom I have given a blood transfusion in the pre-operative phase.

Dehydration is an infrequent problem before operation. More often one sees infants who are somewhat over-hydrated by administration of full-strength physiological saline before their admission to hospital. Where there is clinical evidence of dehydration, I would suggest giving half-strength physiological saline with 2·5% glucose solution by the intravenous route. The amount needed is small; the volume in millilitres is equal to 5% of the infant's body weight expressed in grammes. This fluid is given over a period of three hours, and at the end of this time the clinical state of hydration is assessed. If necessary a further small volume may be given.

Where infection is suspected or known to be present, the parenteral administration of antibiotics is commenced; either penicillin, 50,000 units, with streptomycin, 50 milligrammes, or tetracycline, 50 milligrammes, every six hours. Vitamin K is given also by injection. It is a reasonable precaution that for every newborn baby who is to undergo a serious operation, a blood transfusion should be running when the operation is commenced.

Post-Operative Period.

In the early post-operative phase, most neonates who have had a major surgical operation are shocked. This is due to unavoidable exposure and handling, with at times inadequate replacement of blood loss during the operation. Moreover, while the operation is in progress it is often difficult to recognize that shock is developing.

It is good practice for the physician to be present in the theatre during the operation to assist the anæsthetist in observing the condition of the baby. Even after the operation, shock may not be recognized by those unfamiliar with this problem. A slow heart rate and cold pale skin are frequent signs of shock. If, in addition, the respirations are slow and even irregular, the mortality rate is very high.

Moderate external heating and oxygen administration are indicated. Most patients urgently need a blood transfusion followed by serum. I have frequently given a volume of 50 to 100 millilitres of blood and 50 millilitres of serum to these babies, over a period of three hours, following operation.

The airway, pleural and extra-pleural suction, and gastric suction, require much care in this early period after operation. Posture and frequent oro-pharyngeal suction are usually necessary after the repair of tracheo-œsophageal fistula; occasionally it may be necessary to perform a bronchoscopy as well. The common practice at the Royal Alexandra Hospital for Children is to leave the endotracheal tube in position for twelve hours after the repair of a tracheo-œsophageal fistula; this ensures a good airway and allows easy access for tracheo-bronchial suction. Pleural and extra-pleural suction are frequently unsatisfactory, despite careful attention. Clinical and radiological assessment of the condition of the chest is undertaken early in the post-operative period. When this is necessary, a needle is placed in the pleural or extra-pleural space. When gastric suction is not functioning well the tube is removed, and, as described previously, passed again.

The administration of electrolyte-containing fluids is not an urgent matter. It is frequently necessary to give fluids parenterally for seven days and even longer after the surgical relief of intestinal obstruction. Where only a small fluid maintenance is required for a few days, the subcutaneous route will suffice, but where losses of fluid are occurring as a result of gastric or ileostomy suction, the intravenous route becomes necessary because of the relatively large amounts of fluid involved.

The physician checks personally the fluid balance record for accuracy and detail. If an ileostomy has been performed, it is better to institute ileostomy suction rather than to allow drainage into pads. The baby is weighed daily if it is possible to do so without great disturbance. If laboratory facilities are available, analysis of gastric and ileostomy fluids for electrolyte concentrations, and serum electrolyte studies are undertaken every second day. Our studies have shown that fluid obtained by gastric suction frequently has a composition of the following nature: sodium, 50 to 70 milliequivalents per litre; potassium, 10 to 20 milliequivalents per litre; chloride, 90 to 110 milliequivalents per litre. Enormous variations, however, have been found; for example, one specimen contained 300 milliequivalents of sodium per litre. The concentrations of electrolytes in the serum of normal neonates are rather different from those found in older children and adults. Examples are: potassium, 9·8 milliequivalents per litre at birth, 7·8 milliequivalents per litre at ten days; bicarbonate, 22 milliequivalents per litre at birth (Smith, 1952).

The fluid to be given can be divided into two parts: that necessary for ordinary maintenance needs (that is, the volume a normal baby would be taking by mouth), and that necessary to replace losses of fluid occurring from gastric and ileostomy suction. For maintenance needs I have used equal volumes of a 5% solution of glucose in distilled water and one-quarter strength physiological saline in 3·75% glucose solution. This is, of course, equivalent to one-eighth strength physiological saline. The volume to be administered for maintenance is calculated from the formula: Volume in millilitres = (infant's weight in kilograms) × (number of days of infant's age) × 10. This applies to infants up to fourteen days of age. Where losses of either gastric or ileostomy fluids, or both, are occurring, these losses can be replaced volume for volume as they are occurring. If electrolyte concentrations of these fluids have been determined, then electrolyte losses can be replaced mathematically. If such analysis

cannot be done, then one-half strength physiological saline in 2·5% glucose solution is a satisfactory replacement fluid.

Potassium need not be administered, if only maintenance fluid is being given; but if losses are occurring from the intestinal tract, then five milliequivalents of potassium are given each day. This is administered as potassium chloride solution which has been injected into a flask of half strength physiological saline or other fluid to be given to the patient. (Ampoules of 10 millilitres containing 10 millimols of potassium chloride are available; one millilitre of this solution contains one milliequivalent of potassium.) Protein, as human albumin, is given daily to babies receiving fluids intravenously for more than forty-eight hours. Fifteen millilitres of a 25% albumin solution are injected each day into a flask of fluid to be given to the patient; this supplies one gramme of protein per kilogram of body weight, and also supplies three milliequivalents of sodium.

Alimentary tract feeding is undertaken as early as possible. During repair of a tracheo-œsophageal fistula, a polyethylene tube can be passed down the œsophagus and this allows feeding to commence approximately two days after operation. After the relief of intestinal obstruction, oral feeding is not commenced until it is reasonably certain that the obstruction has been relieved; this usually means that oral feeds are not commenced until the sixth post-operative day or later.

Cortisone has not been used for any of our patients. If it is used, one should remember that sodium retention and a deficient nitrogen balance are usual effects of its administration. I suspect that, if sufficient attention is given to other aspects of management, cortisone is unnecessary.

In conclusion, I have tried to give the impression that the total care of a newborn baby undergoing a major surgical procedure requires the services of a skilled, experienced and enthusiastic pædiatrician, in addition to a surgeon and an anæsthetist of similar calibre. This idea is expressed in a slightly different way by Gross (1953) when introducing this subject in his text-book of pædiatric surgery:

> The most difficult chapter to write is that concerning pre- and post-operative care. It is possible to put in writing certain policies and well defined techniques. . . . However, the care of children requires in addition a certain indefinable something which is well-nigh impossible to describe and transmit to others by spoken or written word.

Summary.

1. The difficulties to be faced are briefly reviewed. These are: (a) associated congenital abnormalities; (b) neonatal physiology: adrenal, renal, temperature control and blood coagulation; (c) impairment of vital capacity; (d) infection; (e) fluid and electrolyte balance; (f) nutrition; (g) prematurity.

2. An outline is drawn of the management, in the pre-operative period, of certain problems, including (a) transport, (b) investigation, (c) decreased vital capacity, (d) oro-pharyngeal secretions in tracheo-œsophageal fistula, (e) gastric suction, (f) hypothermia, (g) shock, (h) dehydration, (i) infection.

3. The most important aspects of the post-operative management are: (a) shock, (b) airway, (c) pleural and extra-pleural suction, (d) gastric suction, (e) fluid and electrolyte administration, (f) oral feeding, (g) cortisone.

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TOPICAL HYDROCORTISONE THERAPY IN DISEASES OF THE SKIN: A CLINICAL EVALUATION.

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AN analysis of 100 cases of diseases of the skin treated with local applications of hydrocortisone ointment in our private practice between June and December, 1954, was made with a view to assessing the results achieved. In this way we hoped to find out the skin diseases in which it was likely to be of benefit, and so conserve supplies and not put our patients to the expense of this costly remedy by prescribing it for those patients who are not likely to be benefited.

For the purpose of this paper certain skin diseases are designated with the aim of making them easily recognizable by the reader, regardless of the finer points of nomenclature. For example, when eczema is designated, it here implies a condition in which we regarded the constitutional factors as being predominant in the ætiology, and when dermatitis is referred to it implies that local contactants were considered to be the predominant feature whether they acted as primary irritants or as sensitizers. Similarly, neurodermatitis here implies a local circumscribed area of lichenification perpetuated by rubbing, with no objective signs or previous history of contact dermatitis; whilst Besnier's prurigo refers to the disseminated form of neurodermatitis seen in the asthma-eczema-prurigo syndrome, and manifested usually by bilaterally symmetrical areas of lichenification involving chiefly the antecubital and popliteal fossæ, but occurring elsewhere in addition to these sites of election. The other conditions referred to, we think, will be easily recognizable without further explanation. There were no controls as such, but most of the patients had failed to respond to other methods of treatment before hydrocortisone was used.

The results of treatment were assessed as follows: "excellent", "good", "fair", "condition unchanged", and "condition aggravated", according to our separate observations. Although this is not scientific, we have tried to be honest and unbiased in our assessments.

Our results coincide with those obtained by Heilesen, Kristjansen and Reymann (1954) in regard to ano-genital eczema, circumscribed neurodermatitis and eczema in which they reported favourable results. The other dermatoses considered in this paper were not reported on by them. Our results also coincide with the results obtained by Malkinson and Wells (1954) in regard to eczema and dermatitis, *pruritus ani* and particularly dermatitis of the eyelids and some cases of diskoid eczema. However, these authors achieved a favourable response in four cases out of seven of chronic itching *otitis externa*, whereas it is our opinion that results of treatment of this condition with hydrocortisone ointment are inferior to those of other methods used by us.

Dermatitis.

Thirty-seven patients with dermatitis due to various causes, some elicited and some not, were treated with hydrocortisone ointment. In 13 of these cases an excellent response was obtained and in 15 a good response was obtained, the condition of five patients was moderately improved, that of four patients remained unchanged, and in no case was the condition aggravated.

In the majority of cases in which the condition improved, good results were obtained within one week, and seldom was it necessary to continue treatment for more than four weeks. In some cases it was found that after the acute stage of the dermatitis had subsided very little additional

TABLE I.

The Results of Treatment with Hydrocortisone Ointment in 100 Cases of Various Dermatoses Considered Under Nine General Headings.

Diagnosis.	Number of Cases.	Excellent Response.	Good Response.	Fair Response.	Condition Unchanged.	Condition Aggravated.
Dermatitis	37	13	15	5	4	0
Eczema	11	4	3	2	2	0
Besnier's prurigo	7	1	4	1	1	0
Neurodermatitis	10	1	4	5	0	0
Diskoid eczema	9	2	1	3	3	0
Otitis externa	4	1	1	2	0	0
Pruritus ani et vulvæ	9	4	2	1	0	2
Intertrigo and scrotal eczema	8	6	0	0	0	2
Miscellaneous	5	2	0	0	3	0
Total	100	34	30	19	13	4

improvement followed the continued use of hydrocortisone ointment; then the use of standard remedies, such as tar pastes, Lassar's paste and superficial X-ray therapy, was needed to complete the cure. However, complete cure was achieved with hydrocortisone ointment alone in some cases, but this was always achieved in less than three weeks.

CASE 1.—A., a male patient, aged forty-two years, reported on July 1, 1954, with dermatitis of the eyelids, which was thought to be due to cement. Hydrocortisone ointment (1%) was prescribed, and when the patient was examined one week later the condition had completely cleared. He was last examined on July 15, 1954, at which time he had had no further trouble.

CASE 16.—B., a female patient, aged forty years, had had dermatitis of the third right interdigital space for four months. Repeated examination of scrapings from the area revealed no mycelia. The condition failed to respond to the application of bland soothing creams and superficial X-ray therapy, and after three weeks of this treatment 1% hydrocortisone ointment was prescribed. One week later the patient's condition was greatly improved, ar 1 this improvement was maintained for three weeks. As the eruption had not completely cleared, *Pix Carbonis Preparata*, one drachm, in zinc paste, one ounce, was prescribed, and superficial X-ray therapy was recommenced. After three weeks of this therapy the eruption had completely gone and no further treatment was given.

CASE 11.—C., a female patient, aged sixty-five years, had suffered intermittently from dermatitis of the fingers of both hands for three years. Examination of the patient revealed an erythemato-squamous eruption of the dorsal surface of the fingers of both hands. Lassar's paste was prescribed and superficial X-ray therapy given, and as the condition did not improve, 1% hydrocortisone ointment was prescribed to be applied three times a day. One week later the dermatitis was unchanged, but it was decided to persist with this line of treatment. However, after a further eleven days no improvement was noticed, and so X-ray therapy was given and a mercury and tar paste was prescribed. Since then the condition has only slowly improved.

CASE 8.—D., a female patient, aged fifty years, presented with erythemato-vesicular dermatitis of the chin of five days' duration. Sedation and 1% hydrocortisone ointment were prescribed, and three days later the condition had almost completely disappeared. The patient then ceased treatment, but presented three weeks later with another attack of the dermatitis, which had commenced a few days after she had ceased applying the hydrocortisone ointment. However, she again quickly responded to hydrocortisone ointment, and this, together with superficial X-ray therapy, continued for three weeks, completely cleared the eruption. When the patient was last examined ten weeks later she had not suffered a recurrence.

Thus it is concluded that hydrocortisone ointment has a definite place in the treatment of dermatitis. However, it must not be assumed that every patient with dermatitis should be treated with hydrocortisone ointment from the start, as most patients will be cured by removal from contact with the primary irritant or sensitizing substance which caused the dermatitis, and by the application of the simpler and cheaper remedies which have been used with success by dermatologists for many years. It is in the "problem cases" when response to routine measures is slow, or in those cases in which the condition completely defies the standard methods that hydrocortisone ointment is indicated. An exception to this may be made in acute dermatitis of the eyelids which was found consistently to respond to this treatment. The acute œdema of the eyelids with its consequent obstruction of vision requires urgent relief, and it is considered that the application of hydrocortisone ointment is the most rapid and effective way of obtaining this relief.

The treatment of dermatitis of the face, eyelids and neck has been uniformly excellent, not one patient having failed to respond rapidly and completely. Not so successful, however, was the treatment of dermatitis of the hands. The hands were the site of the dermatitis in the four cases in which no improvement occurred. This may be due to the thicker *stratum corneum* of the hands, compared with the face, and absorption of the active principle of the hydrocortisone ointment may be delayed or impeded by this layer.

Eczema.

Hydrocortisone ointment was used in the treatment of 11 patients suffering from eczema on various sites, such as the face, arms, legs and body. In four cases excellent improvement was obtained, in three cases good improvement, whilst very little response was observed in two cases and no response in two cases. In no case was the condition aggravated.

It became apparent soon after the start of treatment with hydrocortisone ointment that spectacular results were not being achieved in the treatment of infantile eczema in which an extensive facial eruption was the essential feature. It was in the children aged from one to two years, with patchy eczematous lesions on the face and limbs, that the greatest improvement was obtained.

TABLE II.

Results of Treatment of Dermatitis with Hydrocortisone Ointment According to Regions Affected.

Region Affected.	Number of Cases.	Excellent Response.	Good Response.	Fair Response.	Condition Unchanged.	Condition Aggravated.
Hands	14	3	7	0	4	0
Eyelids and face	10	8	1	1	0	0
Miscellaneous	13	2	7	4	0	0
Total	37	13	15	5	4	0

TABLE III.

Dermatitis of the Hands Treated with Hydrocortisone Ointment: Detailed Analysis of the 14 Cases.

Case Number.	Patient's Sex and Age. (Years.)	Ætiological Factors in Dermatitis.	Duration of Symptoms.	Previous Local Treatment.	Response to Hydrocortisone.	Period Before Response.	Total Duration of Hydrocortisone Treatment.	Final Assessment and Remarks.
11	F., 63	Not discovered.	Intermittent, for many years.	Yes.	Condition unchanged.	——	2 weeks.	No improvement.
12	F., 23	Hairdressing lotion ("cold wave"); hyperhidrosis.	6 months.	Yes.	Condition unchanged.	——	2 weeks.	No improvement.
13	F., 23	(Second attack, see Case 12).	2 weeks.	Yes.	Good.	1 week.	3 weeks.	Eruption completely cleared after 2 weeks' treatment.
14	F., 17	Not discovered.	2 months.	Yes.	Good.	1 week.	6 weeks.	Improved greatly, but never completely clear. Hydrocortisone then alternated with Lassar's paste and X-ray treatment given.
15	F., 18	Not discovered.	5 months.	Yes.	Good.	1 week.	2 weeks.	The addition of hydrocortisone ointment to the therapy of the persistent patches enabled complete cure to be achieved.
16	F., 40	Not discovered.	4 months.	Yes.	Good.	1 week.	3 weeks.	Excellent response. X-ray therapy and tar paste used later to complete the cure.
17	F., 31	Proprietary washing compound.	6 months.	No.	Condition unchanged.	——	——	No improvement. Also slow response to bland remedies and X rays.
18	M., 62	Not discovered.	3 weeks.	Yes.	Excellent.	1 week.	4 weeks.	Excellent response after routine measures had failed. Eruption healed completely with the addition of mercury and tar paste to the therapy.
19	F., 59	Not discovered.	3 months.	Yes.	Good.	1 week.	1 week.	Good response to routine therapy. After an acute relapse, she was quickly returned to her previous good condition with hydrocortisone ointment, and other routine measures were continued.
20	F., 40	Not discovered.	2½ months.	Yes.	Excellent.	1 week.	6 weeks.	Excellent response after only fair response to routine measures.
21	F., 66	Not discovered.	3 months.	Yes.	Good.	1 week.	1 week.	Good improvement, but not cured. X-ray therapy continued to effect a cure.
22	M., 34	Not discovered.	2 years.	No.	Condition unchanged.	——	1 week.	No improvement.
23	F., 31	Not discovered.	3 weeks.	No.	Excellent.	3 days.	1 week.	Greatly improved.
24	F., 51	Plant sensitization.	6 weeks.	Yes.	Good.	3 days.	Still under treatment.	Eruption greatly reduced and irritation relieved.

CASE 46.—Baby E., aged four months, developed eczema of the face three months before her first visit. Her brother had previously had severe infantile eczema. Tar paste was prescribed, with some improvement, but the child relapsed after three weeks' treatment. Hydrocortisone ointment, 1%, was then prescribed, and one week later the face was quite clear. The child has since relapsed, despite continued use of hydrocortisone ointment.

CASE 48.—Baby F., aged six and a half months, had a four months' history of eczema of the face. Tar paste and mild sedation were prescribed, and after initial improvement the baby relapsed. Hydrocortisone ointment, 1%, was then ordered, and in one week the eruption was greatly diminished. This improvement continued and the face became normal. The child did not relapse, even after accidentally developing acute solar dermatitis of the face.

CASE 44.—G., a female patient, aged seventeen months, had an erythematous lichenified eruption mainly on the legs, of twelve months' duration. Hydrocortisone ointment was prescribed and two weeks later there was no improvement. However, when *Pix Carbonis Preparata* in zinc paste was prescribed instead of the hydrocortisone ointment, immediate improvement resulted.

CASE 39.—G., a female patient, aged two years, had a dry eczematous eruption on the face, of twelve months' duration, and which had not been relieved by the application of various creams and pastes. One week after the use of hydrocortisone ointment, the lesions had completely healed.

It is not possible to predict in advance which patient with eczema will be benefited by hydrocortisone ointment. Success has been achieved in varying degrees in all types of eczematous eruptions, but consistent improvement has not occurred. Therefore it would seem that the standard remedies should be used first in the treatment of eczema, and hydrocortisone ointment reserved for those patients who do not respond.

Besnier's Prurigo.

Seven patients with flexural eczema (Besnier's prurigo) were treated with 1% hydrocortisone ointment, and of these one gave an excellent response and four a good response. One patient was slightly improved, and the condition of one was unchanged.

The general impression obtained was that patients with Besnier's prurigo could be greatly relieved, but the disease could not be completely eradicated. Superficial X-ray therapy was required in addition, or sometimes the substitution of tar paste and X-ray therapy. If improvement was to occur, it usually did so within a few days of commencement of treatment with hydrocortisone ointment.

CASE 50.—H., a male patient, aged six years, was suffering from flexural eczema affecting the popliteal and ante-cubital fossæ and groins, of twelve months' duration. A tar paste was prescribed and superficial X-ray therapy (50r weekly) was given to the popliteal fossæ. After two weeks' treatment he was much better, but not entirely free of his eruption. Hydrocortisone ointment (1%) was then prescribed and great improvement occurred after four days. The skin was clinically normal in three weeks, and no recurrence has been reported since he was discharged from hospital seven months ago.

This case illustrated an excellent response to hydrocortisone ointment.

CASE 55.—I., a male patient, aged two years, had suffered from flexural eczema of the wrists and popliteal fossæ since the age of six months. There was a family history of eczema, a brother and two cousins being affected. Hydrocortisone ointment (1%) and sedatives were prescribed, and much improvement was manifest in a week. Treatment was continued for four weeks, during which time no further improvement occurred, and so a tar paste was prescribed and superficial X-ray therapy administered.

This case illustrates a good response, but one in which complete cure was not effected.

CASE 53.—J., a male patient, aged three years, presented with an itchy rash on the flexor surfaces of the forearms and the anterior surfaces of the legs, of seven months' duration. Hydrocortisone ointment (1%) was prescribed, but after this had been applied for two weeks the rash was
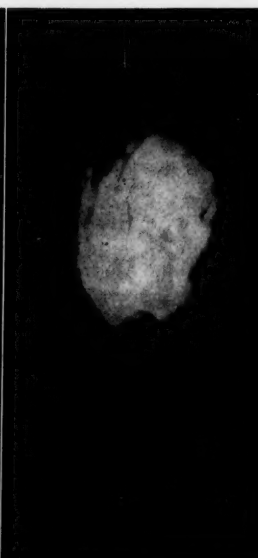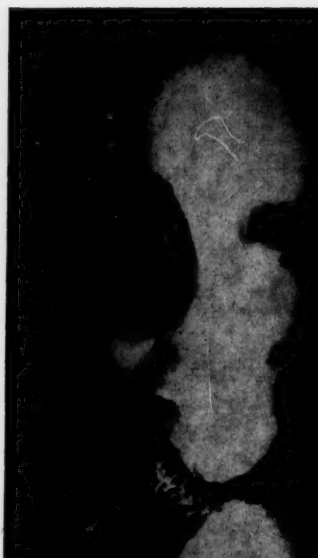
ILLUSTRATIONS TO THE ARTICLE BY C. W. R. PRICE.
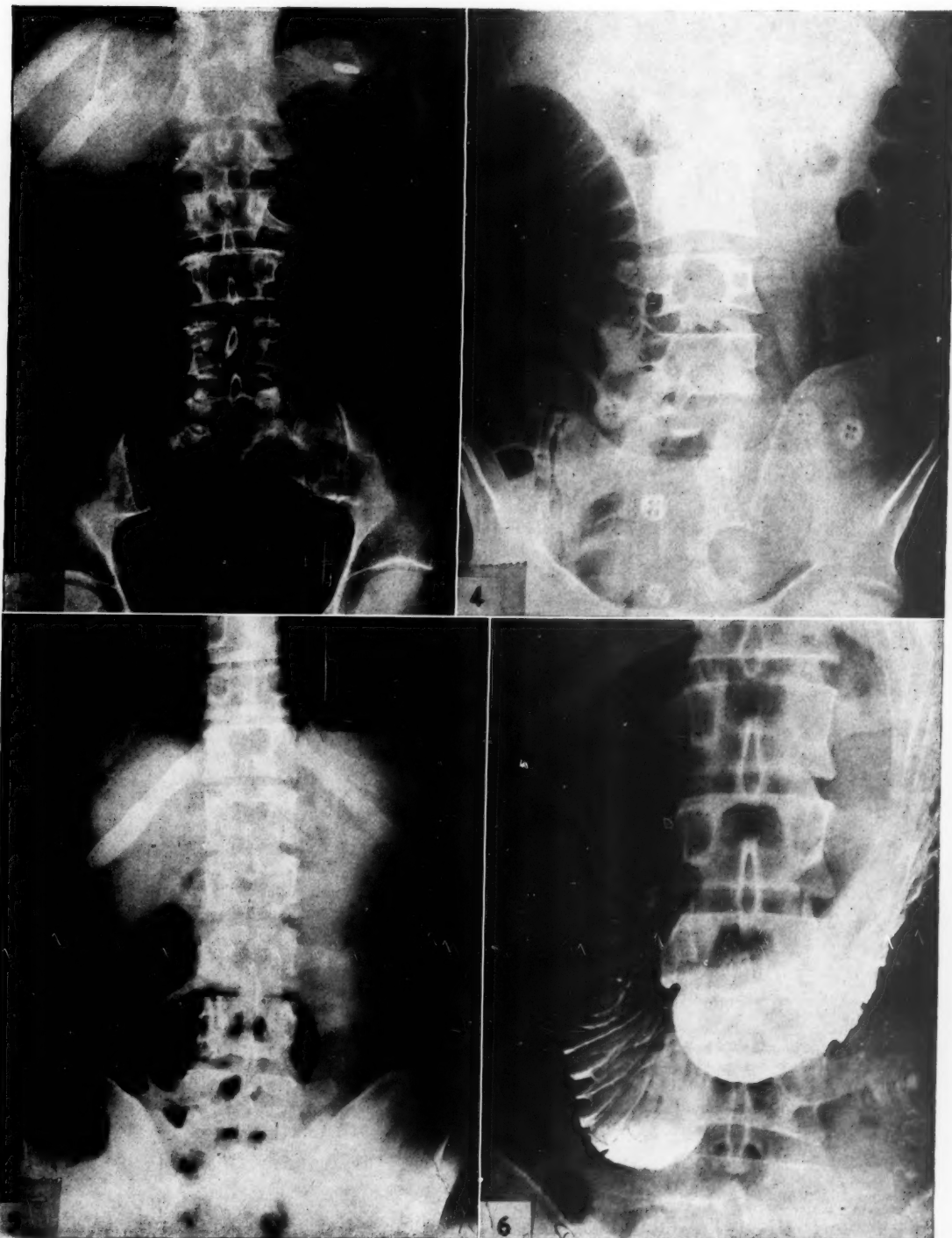
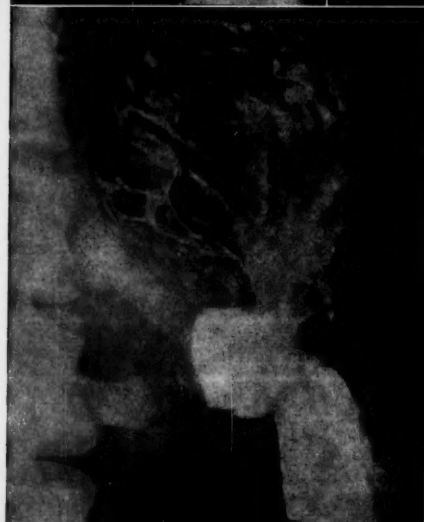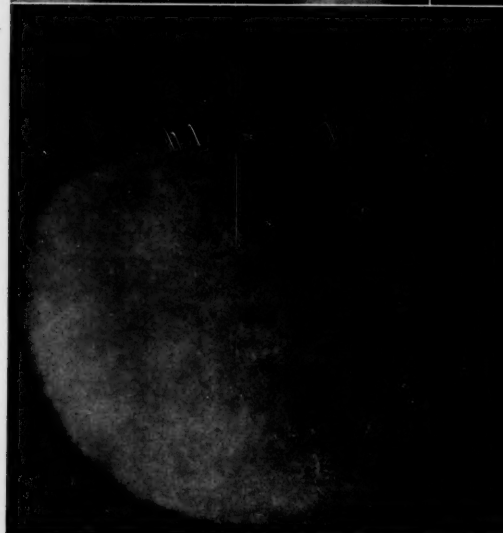ILLUSTRATIONS TO THE ARTICLE BY V. J. KINSELLA.

TABLE IV.

Dermatitis of the Eyelids and Face Treated with Hydrocortisone Ointment: Detailed Analysis of the 10 Cases.

Case Number.	Patient's Sex and Age. (Years.)	Ætiological Factors in Dermatitis.	Duration of Symptoms.	Previous Local Treatment.	Response to Hydrocortisone.	Period Before Response.	Total Duration of Hydrocortisone Treatment.	Final Assessment and Remarks.
1	M., 42	Cement.	2 days.	No.	Excellent.	3 days.	1 week.	Complete cure within one week.
2	M., 76	Pollen sensitization.	4 days.	Yes.	Good.	1 week.	2 weeks.	Improvement, but not cure.
3	M., 54	Dust in wood shed.	18 days.	Yes.	Fair.	4 days.	2 weeks.	Only very slight improvement.
4	F., 56	Face powder.	1 day.	No.	Excellent.	5 days.	1 week.	Quickly cured.
5	F., 70	Not discovered.	4 weeks.	No.	Excellent.	6 days.	3 weeks.	Immediate relief. Also an acute relapse while still under treatment was rapidly cleared up.
6	F., 74	Dry-cleaning agents.	1 month.	No.	Excellent.	1 week.	4 weeks.	Complete cure.
7	F., 74	Dry-cleaning agents.	3 days.	Yes.	Excellent.	1 week.	5 weeks.	A second attack cleared up in a very short time and did not relapse.
8	F., 50	Not discovered.	5 days.	No.	Excellent.	3 days.	6 weeks.	Excellent response, but relapsed as soon as hydrocortisone stopped. Again recovered with hydrocortisone, and this together with X-ray therapy brought about complete cure.
9	F., 40	Wall-flowers.	2 weeks.	Yes.	Excellent.	1 week.	2 weeks.	Dramatic response.
10	F., 51	Not discovered.	4 months.	Yes.	Excellent.	3 days.	Still under treatment. Has been using hydrocortisone for 3 weeks.	Immediate and spectacular improvement. Mild relapse when treatment reduced to every second day, but again improved with daily application.

unchanged. Treatment was therefore changed to the application of tar paste and superficial X-ray therapy, and improvement was slowly achieved.

This is a case in which no response at all occurred with hydrocortisone ointment.

It would therefore seem that the use of hydrocortisone ointment in flexural eczema is useful only as a temporary measure to overcome the initial symptom of itching. When maximal improvement has occurred it may be necessary to resort to tar pastes and superficial X-ray therapy. The ointment may also be useful for patients who do not respond to routine treatment, and for children whose eruptions temporarily relapse owing to extraneous factors such as teething, upper respiratory tract infection *et cetera*.

Neurodermatitis.

Ten patients with neurodermatitis were treated with 1% hydrocortisone ointment. Five patients showed a fair response, which consisted mainly of a reduction in lichenification, but no relief from the symptom of itching. Only

TABLE V.

Dermatitis of Other Sites Treated with Hydrocortisone Ointment: Detailed Analysis of the 13 Cases.

Case Number.	Patient's Sex and Age. (Years.)	Diagnosis.	Ætiological Factors.	Duration of Symptoms.	Previous Local Treatment.	Response to Hydrocortisone.	Period Before Response.	Total Duration of Hydrocortisone Treatment.	Final Assessment and Remarks.
25	F., 9	Dermatitis of arm and leg.	Tincture of iodine and a proprietary iodine ointment.	3 months.	Yes.	Good.	10 days.	4 weeks.	Good, but not a dramatic response.
26	F., 23	Dermatitis of axillæ, neck and arms.	Not discovered.	4 months.	Yes.	Fair.	6 days.	2 weeks.	Moderate improvement. Very little response to any type of treatment, probably owing to emotional instability.
27	M., 53	Dermatitis of lips.	——	2 weeks.	No.	Excellent.	1 week.	1 week.	Eruption completely clear after treatment for one week.
28	F., 42	Dermatitis of neck with dissemination.	Cement.	8 days.	Yes.	Good.	2 days.	2 weeks.	Immediate improvement but additional therapy and X-ray therapy required to complete the cure.
29	M., 63	Dermatitis of leg with dissemination.	A proprietary skin cream applied by the patient.	4 weeks.	Yes.	Good.	1 week.	2 weeks.	Good initial improvement; X-ray therapy and mercury and tar paste needed to complete the cure.
30	M., 28	Dermatitis of leg.	Cutting oils.	1 week.	Yes.	Good.	1 week.	2 weeks.	Very good response.
31	F., 46	Dermatitis of leg with dissemination.	Not discovered.	6 months.	Yes.	Good.	1 week.	4 weeks.	After condition had cleared up with routine therapy, a relapse was rapidly relieved by hydrocortisone ointment. A second relapse some time afterwards rapidly cleared up with hydrocortisone ointment.
32	M., 41	Dermatitis of legs.	Canvas and rubber leggings.	12 months.	Yes.	Good.	1 week.	10 weeks.	Initially a dramatic improvement in the first week in a very chronic case; but improvement was not continued, and the leg remained much the same in spite of continued application.
33	M., 49	Post-traumatic dermatitis (and ulcer of the leg).	Trauma, then irritating local medications.	6 months.	Yes.	Fair.	1 week.	3 weeks.	Little or no improvement.
34	M., 72	Balanitis and dermatitis of penis.	Secondary to extensive dermatitis.	4 months.	Yes.	Excellent.	3 days.	1 week.	Rapidly cleared and did not relapse.
35	M., 40	Dermatitis of penis and scrotum.	Not discovered.	1 week.	Yes.	Fair.	1 week.	1 week.	Some improvement, but no better than with routine measures.
36	M., 56	Balanitis and dermatitis of penis.	Penicillin ointment from local doctor.	11 weeks.	Yes.	Good.	1 week.	2 weeks.	Œdema reduced and eruption much decreased.
37	F., 48	Dermatitis of foot.	Not discovered.	12 months.	No.	Fair.	1 week.	4 weeks.	Some slight improvement only.

TABLE VIII.

Detailed Analysis of 10 Cases of Neurodermatitis Treated with Hydrocortisone Ointment.

Case Number.	Patient's Sex and Age. (Years.)	Site of Lesions.	Duration of Symptoms.	Previous Local Treatment.	Response to Hydrocortisone.	Period Before Response.	Total Duration of Hydrocortisone Treatment.	Final Assessment and Remarks.
56	F., 56	Left leg, right post-axillary fold, right buttock.	1 year.	No.	Good.	5 days.	6 weeks.	Initially good response with reduction of lichenification. Relapsed slightly, and continued use of hydrocortisone ointment *plus* X-ray therapy cured the eruption.
57	F., 38	Legs (anterior aspects of thighs).	8 years.	Yes.	Fair.	1 week.	1 week.	Some reduction of lichenification.
58	F., 41	Right heel.	4 years.	Yes.	Excellent.	1 week.	1 month.	A long-standing troublesome condition was quickly cleared up and did not relapse.
59	F., 20	Nape of neck.	4 to 5 years.	Yes.	Fair to poor.	——	2 weeks.	Lichenification reduced slightly. Itch not relieved. Eruption finally responded to tar paste.
60	M., 62	Anterior surface of leg.	3 years.	Yes.	Fair.	——	2 weeks.	Lichenification and erythema reduced, but symptoms unrelieved.
61	M., 35	Popliteal fossa.	2½ months.	Yes.	Fair.	5 days.	3 weeks.	Some improvement.
62	F., 41	Forearms and hands.	2 months.	Yes.	Fair.	1 week.	3 weeks.	Slight improvement only.
63	M., 25	Feet.	15 months.	Yes.	Good.	——	3 weeks.	Hydrocortisone was used on a persistent patch. When the patient was seen 3 weeks later, the patch had disappeared.
64	F., 35	Foot and leg.	2 years.	No.	Good.	3 days.	Patient still under treatment.	Definite relief from irritation obtained.
65	F., 48	Nape of neck and elbows.	5 months.	No.	Good.	2 days.	Patient still under treatment.	Definite improvement.

no way superior to those obtained with tar paste and superficial X-ray therapy. It is considered that the application of hydrocortisone ointment is indicated as a means of obtaining symptomatic relief and reduction of lichenification in as short a time as possible. Substitution of tar paste and X-ray therapy may be necessary to achieve complete cure. Hydrocortisone ointment is also a useful therapeutic aid when routine therapy fails, or for relapse during routine treatment. Favourable response to hydrocortisone ointment is extremely variable, and relapses occur whilst it is still being used.

Diskoid Eczema.

Nine patients with diskoid (nummular) eczema were treated with 1% hydrocortisone ointment. Two had an excellent response, one a good response, and three a fair response; in three cases no effect was noticed at all. In no case was the condition aggravated. In all cases in which a good response was obtained, the improvement was immediate. It is considered that if no improvement is seen within one week of the commencement of treatment, no benefit is likely to accrue.

CASE 69.—O., a male patient, aged forty-three years, had suffered from recurring attacks of diskoid eczema for the past three years. When examined on June 23, 1954, he presented patches of diskoid eczema on both legs, which had been present for ten days. Hydrocortisone ointment (1%) was prescribed to be used three times a day, and when he was examined five days later his rash was much relieved. On July 5, 1954, his rash had completely disappeared and he was discharged.

CASE 74.—P., a girl, aged twelve years, reported on October 25, 1954, with eczema of the feet and diskoid eczema on the back, of one month's duration. Phenol (1%) in Lassar's paste was prescribed, and this, together with superficial X-ray therapy, relieved the eczema of her feet, but not the eruption on her back. On December 16, 1954, 1% hydrocortisone ointment was prescribed to be applied three times a day to this area. She continued this treatment for five weeks, during which time the eruption became less itchy but remained clinically unchanged. When she was last examined, new diskoid patches were appearing on her abdomen and there was no reduction in the patches on her back.

It was observed that some patients, after an initial good response, relapse despite continued treatment. In one case of this nature the strength of the ointment was increased from 1% to 2·5% and relapse still occurred. It was also observed that after a good response to hydrocortisone oint-

ment, in a subsequent attack it might produce no improvement.

The results of this treatment in diskoid eczema were rather disappointing; only one-third of the patients obtained worthwhile improvement, and the other two-thirds either quickly relapsed or showed no improvement at all.

Otitis Externa.

Four patients with *otitis externa* were treated with 1% hydrocortisone ointment. One gave an excellent response, one a good response, and two a fair response.

In the small number of cases under review we have not been impressed with the value of hydrocortisone ointment in the treatment of *otitis externa*, and consider that consistently better results are achieved with other treatment used by us. Indeed, even in the one case classified as a good response final resort to our routine measures was necessary for complete cure of this patient.

Pruritus Ani et Vulvæ.

Nine patients with *pruritus ani* and *pruritus ani et vulvæ* were treated with 1% hydrocortisone ointment. In four cases there was immediate and complete relief of the pruritus, in two there was much improvement, in one there was some improvement, and in two the condition was aggravated.

CASE 81.—Q., a male patient, aged forty years, presented on June 1, 1954, suffering from *pruritus ani* of eighteen years' duration. He had obtained temporary relief from time to time with local treatment, and had been treated by a psychiatrist for three years. Examination of the patient revealed some radiating folds around the anus, but otherwise the skin was normal. On June 8, 1% hydrocortisone ointment was prescribed to be applied three times a day. Three days later the patient reported that the irritation was much less, and ten days later he said that he was astounded by the improvement and that he had had his first full night's sleep for years. He was given a course of superficial X-ray therapy commencing on June 4, 100r (H.V.L. 0·6) being given weekly to the perianal region. On June 25 he was instructed to use his hydrocortisone ointment at night only, and 1% phenol in zinc cream was prescribed for use in the daytime. On July 9 hydrocortisone ointment was discontinued. Two weeks later there was slight return of the irritation, but this was immediately relieved by infrequent application of hydrocortisone ointment. On September 3 he reported that there had been no pruritus for the previous six weeks, and he was discharged.

TABLE IX.

Detailed Analysis of Nine Cases of Discoid Eczema Treated with Hydrocortisone Ointment.

Case Number.	Patient's Sex and Age. (Years.)	Site of Lesions.	Duration of Symptoms.	Previous Local Treatment.	Response to Hydro-cortisone.	Period Before Response.	Total Duration of Hydro-cortisone Treatment.	Final Assessment and Remarks.
66	M., 22	Arms, legs and face.	12 months.	Yes.	Fair.	1 week.	3 weeks.	Lesions on face cleared up but quickly relapsed whilst the ointment was still being used. Other lesions were unaffected.
67	F., 23	Arms, forearms, chest and abdomen.	3 months.	No.	Condition unchanged.	1 week.	2 weeks.	Entirely without effect.
68	M., 49	Legs.	4 years.	Yes.	Fair.	1 week.	2½ months.	Some improvement, but never completely cured. No greater effect obtained with 2·5% ointment than with 1%.
69	M., 43	Legs.	10 days.	No.	Excellent.	1 week.	2 weeks.	Complete cure in two weeks.
70	F., 45	Legs.	4 months.	Yes.	Fair.	1 week.	1 month.	Some slight improvement.
71	F., 46	Legs.	6 months.	No.	Condition unchanged.	——	——	Patient had previously shown some improvement, but when a recurrence was treated, no effect at all was noticed.
72	M., 55	Legs.	6 months.	Yes.	Excellent.	3 days.	7 weeks.	Patient obtained great relief. Would not use any other medications.
73	F., 42	Hands and legs.	3 months.	Yes.	Good.	1 week.	3 weeks.	Treatment enabled the lesions to commence the healing process after the condition had remained stationary for some weeks.
74	F., 12	Abdomen and neck.	1 month.	Yes.	Condition unchanged.	——	4 weeks.	None of the lesions was relieved and new patches continued to appear.

CASE 79.—R., a male patient, aged fifty years, presented on March 11, 1954, suffering from *pruritus ani* of seven years' duration. Hæmorrhoidectomy had been performed ten months previously with no relief of the irritation. Examination of the patient revealed a lichenified and excoriated perianal eruption. Bland soothing local applications were prescribed, superficial X-ray therapy was administered and sedatives were also prescribed. Improvement followed, but owing to lack of cooperation by the patient relapses occurred from time to time. On June 23 hydrocortisone ointment, which had just become available, was prescribed to be applied three times a day, and he was much improved. The frequency of application was then reduced to once at night. This routine kept him quite free of symptoms, and he was discharged on July 29.

CASE 85.—S., a female patient, aged sixty-nine years, presented on August 31, 1953, suffering from *pruritus ani et vulvæ* of two years' duration. Examination of the patient revealed a bright red abraided eruption in the gluteal cleft and around the vulva. This had been recently aggravated by the application of an anæsthetic ointment. Bland soothing creams and sedatives were prescribed and X-ray therapy was administered, and the patient was discharged on December 10, 1953. In October, 1954, after the death of her husband, her symptoms recurred. Hydrocortisone ointment had been prescribed on September 13, 1954, and when she was examined later she reported that this had aggravated the condition from the first application, and its use was immediately discontinued.

It was observed that excellent results were achieved with the use of 1% hydrocortisone ointment for *pruritus ani et vulvæ*. Six out of the eight patients treated were very greatly improved. The patients were instructed to use the ointment three times a day and when the irritation was relieved to apply it once at night only—at the time when the irritation appears to be worst in most cases. The use of the ointment was combined in some cases with superficial X-ray therapy and sometimes also with bland soothing cream, and all patients were given careful instructions with regard to personal cleanliness, wearing apparel and diet.

Scrotal Eczema and Genito-Crural Intertrigo.

Eight patients with genito-crural and gluteal intertrigo (with or without scrotal eczema in male patients) were treated with 1% hydrocortisone ointment. Six gave an excellent response, and in two the condition was aggravated.

CASE 89.—T., a male patient, aged twenty-six years, had suffered for two years from recurrent scrotal eczema and gluteal intertrigo. Treatment was begun on March 15, 1954, with sedatives, soothing lotions and creams and superficial X-ray therapy to the gluteal cleft; but his condition was aggravated by his occupation as the driver of a bus, in which he sat for long hours with consequent increased perspiration of the affected parts. After some weeks' treatment the affected skin looked normal, although he was still worried by some irritation. On July 9 hydrocortisone ointment (1%) was prescribed, and after one week the irritation had gone. There has been no return of his trouble, and on being discharged he requested a prescription for the ointment to have at hand should his trouble recur, so confident was he in its efficacy.

CASE 93.—U., a male patient, aged thirty-six years, presented suffering from gluteal intertrigo of four and a half months' duration. Hydrocortisone ointment (1%) was prescribed, but was discontinued after the first application, which aggravated the condition. Soothing washes and a bland antipruritic cream were then prescribed, and these, together with superficial X-ray therapy, resulted in rapid improvement.

Most satisfactory results in the treatment of scrotal eczema and intertrigo of the genito-crural and gluteal folds with 1% hydrocortisone ointment were achieved.

TABLE X.

Detailed Analysis of Four Cases of Otitis Externa Treated with Hydrocortisone Ointment.

Case Number.	Patient's Sex and Age. (Years.)	Duration of Symptoms.	Previous Local Treatment.	Response to Hydro-cortisone.	Period Before Response.	Total Duration of Hydro-cortisone Treatment.	Final Assessment and Remarks.
75	F., 51	2 years.	No.	Fair.	Many weeks.	3 months.	Some improvement, but responded better to mercury and tar paste and X-ray therapy.
76	F., 39	4 months.	No.	Excellent.	4 days.	1 week.	Excellent result, and no relapse after cessation of treatment.
77	M., 50	10 years.	Yes.	Fair.	1 week.	3 weeks.	No response.
78	F., 41	8 months.	Yes.	Good.	3 days.	5 months.	An appreciable degree of improvement, but not completely cured until mercury and tar paste were substituted for the hydrocortisone ointment.

TABLE XI.
Detailed Analysis of Nine Cases of Pruritus Ani et Vulvi Treated with Hydrocortisone Ointment.

Case Number.	Patient's Sex and Age. (Years.)	Diagnosis.	Duration of Symptoms.	Previous Local Treatment.	Response to Hydro-cortisone.	Period Before Response.	Total Duration of Hydro-cortisone Treatment.	Final Assessment and Remarks.
79	M., 50	*Pruritus ani.*	6 to 7 years.	Yes.	Excellent.	2 weeks.	5 weeks.	Completely relieved after 2 weeks, and no relapse after cessation of treatment.
80	M., 40	*Pruritus ani.*	18 years.	Yes.	Excellent.	10 days.	3 months.	Excellent improvement in a condition which had persisted for 18 years. Relapsed when treatment ceased, but when last examined, symptoms were controlled by using hydrocortisone ointment at night only.
81	F., 44	*Pruritus ani et vulvœ.*	12 months.	No.	Excellent.	1 week.	4 weeks.	Excellent response.
82	F., 43	*Pruritus vulvœ.*	23 years.	Yes.	Good.	3 days.	3 weeks.	Greatly improved after 3 days' treatment of recurrent *pruritus vulvœ* of many years' standing.
83	F., 31	*Pruritus vulvœ.*	6 weeks.	Yes.	Good.	1 week.	5 weeks.	At first there was much relief, but after 5 weeks' treatment the condition was aggravated.
84	F., 31	Second attack (Case 83).	3 weeks.	Yes.	Condition aggravated.	——	1 week.	
85	F., 69	*Pruritus ani et vulvœ.*	2 years.	No.	Condition aggravated.	Response immediate.	——	Aggravation of the condition from the first application.
86	F., 38	*Pruritus ani et vulvœ.*	4 years.	Yes.	Fair.	1 week.	3 weeks.	Slight improvement, but later caused stinging.
87	F., 41	*Pruritus ani et vulvœ.*	8 months.	Yes.	Excellent.	1 week.	3 months.	Excellent response. Required very little maintenance therapy, an occasional application of the ointment sufficing.

TABLE XII.
Detailed Analysis of Eight Cases of Intertrigo and Scrotal Eczema Treated with Hydrocortisone Ointment.

Case Number.	Patient's Sex and Age. (Years.)	Diagnosis.	Duration of Symptoms.	Previous Local Treatment.	Response to Hydro-cortisone.	Period Before Response.	Total Duration of Hydro-cortisone Treatment.	Final Assessment and Remarks.
88	M., 30	Intertrigo and scrotal eczema.	5 years.	No.	Excellent.	5 days.	1 month.	Immediate and continued relief.
89	M., 26	Intertrigo and scrotal eczema.	2 years.	Yes.	Excellent.	1 week.	6 weeks.	After only fair response to routine measures eruption cleared rapidly and completely with hydrocortisone ointment.
90	M., 42	Genito-crural inter-trigo.	10 years.	Yes.	Excellent.	3 days.	3 weeks.	Excellent and rapid response. No relapse.
91	M., 50	Intertrigo and scrotal eczema.	10 years.	Yes.	Excellent.	1 week.	1 month.	Rash completely cleared with hydrocortisone ointment and X-ray therapy, after no relief had been obtained with routine measures.
92	M., 45	Genito-crural inter-trigo.	4 months.	Yes.	Condition aggravated.	——	——	Worse after 9 days' treatment. Emotionally unstable patient.
93	M., 36	Gluteal intertrigo.	4½ months.	No.	Condition aggravated.	——	——	Aggravated by first application, use immediately discontinued.
94	M., 58	Gluteal and genito-crural intertrigo.	4 weeks.	No.	Excellent.	3 days.	1 month.	Excellent and rapid response.
95	F., 54	Intertrigo of gluteal cleft and vulva.	4 months.	Yes.	Excellent.	1 week.	1 month.	A relapse, vagina habing been painted for a discharge. Immediate and complete cure with hydrocortisone ointment.

TABLE XIII.
Detailed Analysis of Five Cases of Miscellaneous Conditions Treated with Hydrocortisone Ointment.

Case Number.	Patient's Sex and Age. (Years.)	Diagnosis.	Site of Lesions.	Duration of Symptoms.	Previous Local Treatment.	Response to Hydro-cortisone.	Period Before Response.	Duration of Hydro-cortisone Treatment.	Final Assessment and Remarks.
96	F., 50	Morphœa.	Leg.	2 years.	No.	Condition unchanged.	——	2 months.	No definite improvement seen.
97	M., 48	D.D.T. eruption.	Legs and lower part of abdomen.	12 months.	Yes.	Condition unchanged.	——	1 week.	No improvement.
98	F., 25	*Erythema multiforme.*	Lips.	2 weeks.	Yes.	Excellent.	3 days.	2 weeks.	Crusted ulcerated lips were greatly relieved in 3 days and normal in 17 days.
99	M., 12 weeks	Napkin erythema.	——	8 weeks.	Yes.	Condition unchanged.	——	1 week.	No improvement.
100	F., 2	Papular urticaria.	Arms and scapular regions.	12 months.	Yes.	Excellent.	5 days.	2 weeks.	New lesions of papular urticaria cleared up in less than 1 week after proving resistant to previous methods of treatment for 2 to 4 weeks.

Response to treatment was rapid, occurring within a few days. Although in two of the cases the condition was aggravated, it was considered that in one this may have been due to uncleanliness and to the poor mentality of the patient.

Miscellaneous Dermatoses.

In Table XIII are shown five cases of miscellaneous dermatoses that could not be classified with the other cases described. It was not surprising to find no improvement in the case of morphœa (localized scleroderma) or in the

purpuric eruption, but it was surprising to find no improvement in the single case of Jacquet's napkin erythema, as this was in the first stage of erythema without any ulceration.

It was pleasing to find an excellent and rapid response in the patient suffering from papular urticaria, which is usually a very refractory condition to treat by any other method.

Quite the most gratifying result was that achieved in treating the lesions of the lips in the patient suffering from *erythema multiforme*. This patient was in great discomfort and distress, and was completely cured of the mouth lesions in three days.

Summary and Conclusions.

An analysis has been made of the treatment with hydrocortisone ointment of 100 patients with diseases of the skin. These have been considered under the headings of nine different dermatoses, the designations used being for simplicity in understanding the diseases under discussion, and not with attention to the finer and controversial points in dermatological nomenclature.

In certain dermatoses an analysis has been made under regional headings as well as general headings, as response seems to vary at different sites.

Failure to respond to routine treatment other than the application of hydrocortisone ointment has been used as a criterion in most of the cases instead of deliberately applied controls.

Hydrocortisone ointment has a definite place in the treatment of skin diseases. It should not be used indiscriminately, as results are negative or inferior to other methods in many diseases of the skin.

It should not be used in the presence of infection of the skin, or at any rate unless it is combined with some anti-infective measure with which it is compatible.

The best results were obtained in the treatment of the following conditions: *erythema multiforme* involving the lips, papular urticaria, dermatitis and eczema of the face, especially the eyelids, eczema of the scrotum, *pruritus ani et vulvæ* and intertrigo of the genito-crural and gluteal folds.

Results were disappointing in Besnier's prurigo, diskoid eczema and *otitis externa*. In four patients, two suffering from scrotal eczema and two suffering from *pruritus ani* and intertrigo of the genito-crural and gluteal folds, the condition was aggravated.

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Reports of Cases.

ACUTE INVERSION OF THE UTERUS SUCCESSFULLY TREATED BY THE HYDROSTATIC METHOD: REPORT OF A CASE.

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ACUTE inversion of the uterus is a rare and grave complication of pregnancy. Five cases have occurred in 81,140 deliveries at the Royal Women's Hospital, Melbourne, during the last sixteen years. Since the last instance, there have been 30,000 deliveries. There was one maternal death resulting from this complication. During this time, 12 additional patients have been admitted to this hospital already suffering from acute inversion of the uterus, and one of these also died. In 1945, a new and simple technique for the replacement of the acutely inverted uterus was recorded (O'Sullivan, 1945). This procedure has since become known as the hydrostatic method. There have now been many reports of its successful employment. However, there are in the current textbooks of obstetrics few full descriptions of the technique which, it is considered, should be widely known, because acute inversion of the uterus presents as an emergency in which the patient's life is threatened.

Clinical Record.

Mrs. W., a *primipara*, aged twenty-three years, was transferred from a private hospital to the Royal Women's Hospital, Melbourne, where she was admitted at 2.30 a.m. on April 1, 1955. The following history was obtained from the patient's personal medical attendant, who had requested her admission to hospital.

The course of pregnancy had been uneventful. The estimated date of confinement was March 18, 1955. Two medicinal stimulations with pituitrin had been administered during the last week of March. Both were unsuccessful. On the morning of March 31, the patient's blood pressure was 140 millimetres of mercury, systolic, and 100 millimetres, diastolic, and her urine, by the heat and acetic acid test, contained "one-quarter" albumin. There was no clinical evidence of œdema. A diagnosis of preeclampsia was made, and at 1 p.m. surgical induction of labour by artificial rupture of the membranes was performed. At 3 p.m. labour commenced, and at 11.52 p.m. a normal delivery of a living female baby, weighing seven pounds eight ounces, was accomplished. Immediately after delivery, ergometrine maleate, 0·125 milligramme, was given by intravenous injection, and ten minutes later the placenta was readily expressed by the Dublin method. There was no traction on the umbilical cord. The blood loss during the third stage was considered to be of average amount. One-quarter of an hour later the patient's condition was observed to be satisfactory. At 12.30 a.m. on April 1, post-partum hæmorrhage commenced; the pulse rate was then 110 per minute. Ergometrine maleate, 0·125 milligramme by intravenous injection, and pituitrin, two units by intramuscular injection, were given. The hæmorrhage then ceased. The total blood loss was estimated to be not more than 40 ounces. The patient's general condition now further deteriorated, the pulse rate rising and the pulse volume decreasing. At 12.50 a.m., pelvic examination revealed inversion of the upper two-thirds of the uterus into the lower one-third of the uterus and vagina. The patient was given morphine sulphate, 10 milligrammes by intramuscular injection, an intravenous infusion of dextrose (5%) in normal saline was commenced, and transfer to the Royal Women's Hospital was arranged.

On examination, after admission to the Royal Women's Hospital, the patient was found to be in a state of severe shock. She was very pale. The pulse rate was 150 per minute and the volume poor. The blood pressure was 80 millimetres of mercury, systolic, and 60 millimetres, diastolic. She did not complain of pain. Palpation of the abdomen revealed a tender central mass rising out of the pelvis to a level approximately midway between the *symphysis pubis* and the umbilicus. This mass resembled the normal puerperal involuting uterus in shape, although it was somewhat broader. An indentation, the inversion funnel, could be felt at the centre of its upper margin. There was no hæmorrhage *per vaginam*. Catheterization of the bladder yielded three ounces of urine, and this, on being tested, contained "one-half" albumin.

The foot of the bed was elevated and a blood transfusion commenced. The administration of penicillin and sulphacetamide, by intramuscular injection, was commenced as prophylactic treatment against infection.

By 4 a.m., one and one-half hours after her admission to hospital, the patient's general condition had greatly improved. The pulse rate was 108 per minute, and the blood pressure was 90 millimetres of mercury, systolic, and 60 millimetres, diastolic. The transfusion of two pints of blood had been completed. At 4.30 a.m., there was no

evidence of further improvement in the patient's condition despite an additional transfusion of one pint of blood. It was now decided to attempt reposition of the uterus by the hydrostatic method.

It was thought that caudal anæsthesia might be of advantage, in that it might reduce efferent sensory impulses from the pelvic viscera which could cause or be causing shock. After 30 millilitres of "Xylocaine" (1% solution) had been injected, the patient suffered a very brief generalized convulsion. Whether this convulsion was due to the injection of the caudal anæsthetic, or whether, in view of the history of preeclampsia, this was an eclamptic fit, is debatable. General anæsthesia was immediately induced with nitrous oxide and oxygen, and this was later supplemented with a little ether.

The patient was then placed in the lithotomy position, and after full antiseptic preparation of the operative field, a bimanual pelvic examination was performed.

This showed that there was a complete inversion of the uterus. The apex of this soft, dark purplish-red, slightly oozing mass extended down almost to the vaginal introitus. The area of placental attachment was identified as being at the fundus and extending on to the posterior uterine wall. The inversion funnel could be readily felt on combined abdominal and pelvic palpation.

Preparations for replacement of the uterus by the hydrostatic method were now completed. Six litres of sterile normal saline had been warmed to a temperature of 120° F. A sterile douche can was suspended three feet above the vaginal introitus and then filled with this saline solution. The rubber tubing from the douche can was led so that its distal end was in the region of the posterior vaginal fornix. The patient was placed in Trendelenburg's position and the vagina was gradually filled with the saline solution. In this way, it was hoped to obviate the risk of air embolism. Once the vagina was filled, the introitus was plugged by the fist of the hand holding the rubber tubing in the vagina, and an assistant brought the labia together about the wrist. The saline solution was now allowed to run freely.

A remarkable degree of ballooning of the vagina followed, and, on abdominal palpation, the inversion funnel could be felt to be progressively dilating. At the same time the uterus gradually rose in the vagina. After approximately ten minutes, it was found that reposition, except for a fundal dimple, had been achieved. This fundal dimple was very easily corrected by the hand which had now been introduced into the uterine cavity. The correction would almost certainly have occurred if the hydrostatic pressure had been further maintained. Examination revealed that the uterine wall over the placental site at the fundus was abnormally thin. Ergometrine maleate, 0·5 milligramme, was given by intravenous injection, and the uterus was felt to contract and retract with no tendency for the inversion to recur.

Once reposition had been achieved, the patient's condition showed an immediate improvement. The blood pressure rose to 110 millimetres of mercury, systolic, and 70 millimetres, diastolic. The pulse rate fell to 84 per minute. Two hours later, the systolic blood pressure had risen to 125 millimetres of mercury, at which level it persisted.

In order to reduce the hazard of pelvic vein thrombosis and possible embolism, anticoagulant therapy was commenced. The oral administration of ethylidine dicoumarol was controlled by regular estimations of the prothrombin concentration.

The puerperium was quite uneventful. Uterine involution proceeded at a normal rate, and the lochia was not excessive. On the second day the urine was free from albumin, and a hæmoglobin estimation showed the value to be 9·5 grammes per 100 millilitres. A blood transfusion of two pints was given. On the fourth day, the patient was allowed to sit out of bed.

Comment.

The occurrence of acute inversion of the uterus must always be considered when any patient develops shock after delivery. The entity of spontaneous inversion is well recognized, although mismanagement in the delivery or attempted delivery of the placenta is the usual cause of this complication. It is likely that the incidence will be reduced by the common present-day practice of giving ergometrine or posterior pituitary extract at the time of or immediately after delivery, and so causing an almost instantaneous firm contraction and retraction of the uterus.

The inversion is almost invariably complete (O'Sullivan, 1955). There may be present with a complete inversion, as is illustrated in this case, a central lower abdominal tumour formed by the inversion funnel and its contents. This resembles the post-partum uterus, and an error in diagnosis may result if examination is careless and incomplete.

A great number of methods have been described for the treatment of acute inversion (Fenton and Singh, 1950). The hydrostatic method of replacement of the uterus offers a simple and gentle technique for the management of this complication. There is no possibility of rupture of the uterus as in forcible manual reposition.

For hydrostatic replacement an antiseptic solution is usually advised (O'Sullivan, 1955). It is believed that a sterile normal saline solution is safer, in that, if any fluid should gain entry to the circulation or find its way along the Fallopian tubes to the peritoneal cavity, there is less danger to the patient. Prophylactic measures against infection, air embolism and pelvic vein thrombosis should be taken, and these have been outlined in the case report.

It is of interest to note that, in 1930, the aunt of this patient was admitted to the Royal Women's Hospital, Melbourne, suffering from acute puerperal inversion of the uterus. The treatment was hysterectomy.

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O'SULLIVAN, J. V. (1945), "Acute Inversion of the Uterus", *Brit. M. J.*, 2: 282.

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Reviews.

Medical Emergencies: Diagnosis and Treatment. By Francis D. Murphy, M.D., F.A.C.P., and Associate Authors; foreword by George Morris Piersol, M.D.; Fifth Edition, 1955. Philadelphia: F. A. Davis Company, Publishers. Sydney: Angus and Robertson, Limited. 9" × 6", pp. 622, with many illustrations. Price: 82s. 6d.

Now in its fifth edition, this book illustrates the rapidly changing face of therapeutic medicine by appearing within three years of the last edition. The enormous advance in this field has forced Professor Murphy to enlist the assistance of five colleagues of the medical staff in the Department of Medicine at Marquette University and the Milwaukee County Hospital.

As previously, the title is misleading. The bulk of the 600 pages deals with acute conditions, including three chapters on blood dyscrasias, three on acute infections and two on tropical diseases, hardly subjects in which are found many emergencies in the accepted sense of the word. However, once understanding of the book's real intention is gained, one finds a valuable fund of information which will render good service to all who study and practise general medicine. With the accent on diagnosis, differential diagnosis and management, this edition retains the impression of reliability based on tried and proven measures in the hands of the authors. The reader is led point by point through examination and then treatment of the patient in the urgent, acute, and not-quite-so-acute conditions which beset mankind.

Arranged largely according to the systems, there are also useful chapters on acute abdominal conditions, infections, tropical diseases, poisoning and drug therapy. It is

pleasing to find expansion of the section on the nervous system, particularly dealing with convulsions and the psychoses.

There are numerous helpful illustrations and an index.

British Obstetric and Gynæcological Practice: 1955. London: William Heinemann (Medical Books), Limited. "Obstetrics", edited by Eardley Holland, M.D. (London), F.R.C.P., F.R.C.S., F.R.C.O.G. 10″ × 7½″, pp. 1180, with 395 illustrations. Price: 115s. "Gynæcology", edited by Aleck Bourne, M.A., M.B., B.Ch. (Cantab.), F.R.C.S., F.R.C.O.G. 10″ × 7½″, pp. 854, with 363 illustrations. Price: 95s.

IN this comprehensive book, the volume on obstetrics embodies the contributions of thirty-eight well-known authors and teachers from the British Isles and Australia in a first quality text-book which fairly represents the British concept of modern obstetrics, as indicated by the title. Not all the contributors are obstetricians. Physicians, pædiatricians, anatomists, a psychiatrist, a venereologist, a statistician and a barrister play a part, as indeed they do in the problems which so often confront us. Thus the scope passes beyond the field of pure obstetrics to include associated matters of relevance and importance. Too large for the undergraduate, this is an excellent book of reference for the practitioner in obstetrics and for the graduate who studies for a higher degree. Though theories and dogmas are included, the work is essentially devoted to clinical and practical details—a feature which should appeal to those who seek authoritative guidance in their daily work. In spite of the many sources from which it is compiled, it is surprising that one is scarcely aware of any abrupt changes in style, even though the editor himself makes reference to this problem. The paper and print are good, illustrations abundant and the bibliography is extensive; it will surely be approved as a worthy asset by all who use it.

In the volume on gynæcology, companion to the one on obstetrics, Aleck Bourne, one of the most famous names in this field, endeavours to present as far as possible to the reader an up-to-date review of this branch of medicine as practised in Great Britain. In this he is assisted by twenty other contributors, many world famous, drawn from most of the teaching schools of that country. The book thus becomes a mirror of British teaching and practice, methods and technique, as they exist today. At the same time an effort has been made, and one thinks successfully, to preserve a general unity and orderly sequence in the arrangement of the subject matter adequately covering all aspects of gynæcology. The book being essentially of a practical nature, all descriptions of physiology and pathology are restricted to what is necessary for the proper understanding of the clinical viewpoint, and it is well illustrated by original drawings, photographs, diagrams and coloured reproductions. Furthermore, although it contains much sound advice on surgical procedure, technique, difficulties and dangers, the book does not claim to be a manual of operative gynæcology. There is a comprehensive bibliography at the end of each chapter, and the subjects—contentious ones particularly so—are very soundly reviewed and assessed in the light of all the latest research, although, as one would expect, the general tone throughout is conservative. The volume concludes with useful chapters, usually omitted in similar works, on the psychiatric and medico-legal aspects of gynæcology.

To gynæcologists in Australia this volume will represent the core of current British gynæcological teaching and should be read by all who are interested in the specialty. The specialist will find it more of an academical brush-up, but for the general practitioner it should prove an invaluable addition to his reference library.

1955 Medical Progress: A Review of Medical Advances during 1954. Edited by Morris Fishbein, M.D.; 1955. New York: McGraw-Hill Book Company, Incorporated. 9″ × 6″, pp. 358. Price: $5.00.

THIS book is the third in a series which began in 1953; it is again edited by Dr. Morris Fishbein. It endeavours to review progress in medical science during the year 1954, and covers medicine, surgery, pædiatrics, obstetrics and gynæcology, orthopædic surgery and dermatology. Most of the book, however, is devoted to medicine.

Some of the sections are well written, in particular Joslin's admirable review of *diabetes mellitus* and the discussion on serum lipoproteins in coronary disease and atherosclerosis in the chapter devoted to nutrition. Popper and Schaffner write a good account on liver disease, and Boland's section on the rheumatic diseases is informative. One of the best chapters is that on physical medicine.

Other sections are disappointing, tending to consist of informative reports on published work, but without the necessary critical analysis as to its worth. Sometimes an author will refer only to work within the United States.

The main object of an annual review of medical progress is to present new ideas, new therapeutic agents, to present trends of thought and indicate the main lines on which medical research is proceeding. These broad objectives are adequately covered in this book, and for this reason it is well worth reading in full.

Textbook of Gynæcology. By J. H. Peel, M.A., B.M., B.Ch. (Oxon.), F.R.C.S., F.R.C.O.G.; Fourth Edition; 1955. London: William Heinemann (Medical Books), Limited. 8½″ × 5½″, pp. 506, with 206 illustrations. Price: 27s. 6d.

THE fourth edition of a "Textbook of Gynæcology", as is usual with a text-book, opens with a chapter on anatomy. In this section more diagrams could have been included to illustrate details of the pelvic fascial attachments described in the text, as these descriptions are rather involved and require more elucidation.

Generally speaking, however, very little can be said to detract from the merit of this book. It is sound and matter of fact, generally conservative and orthodox in its teaching and expressed opinions, which differ very little from those of Australian medical schools. Discussion of theories is reduced to a minimum, and the dogmatic and didactic presentation of the subject matter makes it ideal for student assimilation. The general layout is excellent, and the appendix of endocrine products with their trade names is again included. The book concludes with chapters on general therapeutic measures, including contraception, physiotherapy, radium and X-ray therapy, pre-operative and post-operative management, and finally one on gynæcological operations quite adequate for those not concerned immediately with minute details of operative technique.

The book is intended primarily for undergraduate students, but the busy general practitioner would also find it handy as a quick reference book. A student cannot draw on personal experience to form opinions of his own. He requires a firm base as a starting point, "to hang his hat on" as it were. Later on he can permit himself the luxury of debating controversial theories or even of contributing something from his own experience. But for examination purposes he must have dogma, clearly presented, concise and up to date, and he certainly gets it in this book.

Books Received.

[The mention of a book in this column does not imply that no review will appear in a subsequent issue.]

"Antibiotics in Nutrition", by Thomas H. Jukes, Ph.D.; 1955; with a foreword by Henry Welch, Ph.D., and Félix Marti-Ibáñez, M.D. New York: Medical Encyclopedia, Incorporated. 9″ × 6″, pp. 127. Price: $4.00.

This book is Number 4 of the series of "Antibiotics Monographs".

"Pertussis in Infancy as a Cause of Behaviour Disorders in Children", by Anna-Lisa Annell, with Statistical Analysis by Elvir Lander, translated from the Swedish by Erica Odelberg; 1953. Uppsala: Almqvist and Wiksells Boktryckeri Aktiebolag. 9″ × 6″, pp. 222.

Based on the results of an investigation carried out at the Department of Child Psychiatry of the University Hospital of Uppsala in collaboration with the Psychiatric Clinic of the same hospital.

"Body Fluids in Surgery", by A. W. Wilkinson, Ch.M., F.R.C.S.E.; 1955. Edinburgh and London: E. and S. Livingstone, Limited. 8½″ × 5½″, pp. 221. Price: 16s.

This is an attempt "to provide . . . first, sufficient information for a basic understanding of the behaviour of the body fluids in health and disease, and secondly, an account of the management of the disturbances of the body fluids which occur in surgical patients".

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PREVENTIVE MEDICINE AND THE GENERAL PRACTITIONER.

PREVENTIVE MEDICINE has gone through many phases. The first trend was an evolution from personal measures to departmental public health control—from propitiation of the gods and the wearing of charms and amulets, the sniffing of pomanders and subjective quarantine, to objective quarantine and, finally, sanitation of the environment. At this stage the gap between preventive and therapeutic medicine was so wide that few practising doctors even realized that there was a gap, or anything at its other side; public health medical officers were a caste of untouchables with duties vaguely connected with drains. This was the stage of the control of the infectious and epidemic diseases. With the introduction of vaccination the pendulum commenced its swing back from environment to individual, and although public health is still under departmental control, and ever will be, preventive medicine has become also part of every doctor's work, as a responsibility to protect his individual patients from possible future disease—not just from diphtheria, pertussis and tetanus by injecting triple antigen, but from threatened hypertension, rheumatoid deformity *et cetera*—and more. This, as will be shown, is a stage, not only of preventive medicine but of medicine as a whole, which is of great and overwhelming importance; we are at the threshold of the development of this newest phase of pre-

ventive medicine, whose ultimate benefit to humanity will be infinitely greater than any of the small things, such as the final conquest of poliomyelitis or malaria, or even cancer, which occupy the interests of the profession at present.

This reference has been inspired by a book—not by the book itself, though it is quite a good one—but rather by what it portends. "The Prevention of Disease in Everyday Practice"[1] consists of a series of monographs by 47 leading workers in various fields; these monographs have been written from the aspect of this extended conception of preventive medicine. Thus E. L. Wynder discusses the intrinsic and extrinsic carcinogens, and measures to prevent their attacking lungs, larynx, alimentary and genito-urinary tracts, skin *et cetera*. E. Press considers the prevention of poisoning; ordinary sensible precautions would prevent most accidental poisonings, and there are often warning signs, such as depression and desperation, which the family physician should recognize in time to take measures to forestall suicidal poisoning. In "Preventive Pediatrics", G. J. Ginandes covers the field from pre-natal care to school age protection—he even commences earlier, with some mention of preventive genetics. No useful purpose would be served by detailing all the other sections of this book, which range from "Preventive Ophthalmology" to "Preventive Proctology", but it should be said that in each section is matter, presented often from a novel angle, that must interest most practitioners. This is a sign of the times, but only one of many. B. Pasamanick and A. M. Lilienfeld[2] have made a survey of a series of mentally deficient persons from the point of view of any abnormalities which they or their mothers might have suffered pre-natally or paranatally, or which they themselves may have suffered neonatally. These authors conclude that a relationship appears to exist between certain abnormal conditions associated with child-bearing and the subsequent development of mental defect in the offspring. The pattern of factors, such as complications of pregnancy, prematurity and neonatal abnormalities, that appears to be associated with mental defect is similar to that of factors previously found associated with stillbirths, neonatal deaths, cerebral palsy, epilepsy and childhood behaviour disorder. The impression given by this paper is that much mental deficiency is not hereditary, but congenital; that familial frequency of mental deficiency can often be traced to genes responsible for small pelves in the females of a family, rather than to genes responsible for retarding mental development.

At the other end of life we are told that one in every 17 of such and such a country's population is now aged sixty-five or more, and are threatened that in ten years' time the figure will be one in 15. The economic complications that have developed because of this shift in the age-balance of population are becoming serious; the thoughtless and the selfish are asking why doctors are allowed to keep alive so many people who would be better dead. There is a spate of books, articles, and even whole journals devoted to geriatrics. Hospitals are finding it more difficult to provide accommodation and nursing care for aged patients,

[1] "The Prevention of Disease in Everyday Practice", by Isadore Givner, B.S., M.D., F.A.C.S., and Maurice Bruger, M.Sc., M.D., C.M., F.A.C.P.; 1955 St. Louis: The C. V. Mosby Company. Melbourne: W. Ramsay (Surgical), Limited. 10″ × 7″, pp. 966, with 50 illustrations. Price: £11.
[2] *J.A.M.A.*, September 17, 1955.

and families are feeling the burden of longer-living grandparents. Industry is torn between demands to raise retiring ages and the claims of the younger workers for more chances of advancement dependent on the retirement of their elders. Governments are already embarrassed by the need for providing more pensions at the expense of a diminishing proportion of actual taxpayers; there are even indications that the burden of aged relatives is forcing the limiting of families, thus threatening to widen the discrepancy between taxpayers and pensioners at an accelerated rate. These are big and important problems, which are increasing by geometrical progression. They threaten the stability of our civilized economy, and they far outweigh the smaller medical problems instanced earlier.

However, many workers are producing data on the same lines as those reported by Pasamanick and Lilienfeld—data which reveal the ever-widening scope for the exercise of preventive medicine in its newest phase—and it is in this phase of personal preventive medicine that the solution of the problems which have been outlined lies. For some decades to come, the aged persons who survive will be those who have been salvaged by new methods, and many of them will be infirm, dependent and a burden. Thereafter, with increasing rapidity, the effects of individual preventive medicine will appear, and there will be among the aged more and more who have not needed to be salvaged from the effects of degenerative disease, because they will have had an even better start in life, as regards health, than the present generation, and their whole lives will have had a healthier course than have our present older generations, so that their old age will be relatively vigorous and independent. It is this new scope of preventive medicine which ensures that the problem of the aged is only a temporary one, which will be solved automatically.

This automatic solution, however, must depend upon the solving of a problem which exists within medicine itself. There is reported to be a world-wide shortage of general practitioners. It is the family doctor, with his long contact with and intimate knowledge of his patients, with his personal interest in them and his unique position of guide and adviser, who is best able to carry out the details of the new, personal preventive medicine. The specialist is almost out of the picture; by the time he sees patients, some damage has usually been done, and his part in preventive medicine is to limit that damage to its existing degree. He is rarely in a position to foresee and forestall damage, as the family physician is. Even if there is a shortage of general practitioners, the remedy is to hand. The time spent in giving triple antigen to a hundred children is less than the time required to treat one child suffering from diphtheria. This principle applies throughout the whole of preventive medicine, so that in the long run the doctor who practises preventive medicine properly has less work to do, as regards both time and effort, than the one who sits in his consulting room waiting for people to become sick enough to need his services. It is often alleged that the Chinese pay their doctor when they are well, but not when they are sick. Such a system might not be a bad one.

Current Comment.

JACQUES BÉNIGNE WINSLOW.

MANY famous people have for one reason or another left the land of their birth and done their life's work in another. Two who come to mind are Frédéric-François Chopin, born in Poland, who achieved fame as a pianist and composer in France, and Joseph Conrad, also born in Poland, who won for himself an unrivalled place in English letters, particularly by his powerful stories of life at sea. R. M. Garraud[1] came upon a copy of the 1776 edition of *L'Exposition anatomique de la structure du corps humain*, and this led him to put together a short account of its author, Jacques Bénigne Winslow, whom he described as "a builder of Franco-Danish good-will". Winslow was born at Odense in 1669. His father was a Lutheran clergyman, whose real name was Mansen; but he was better known by the name of Winslow, which derived from Winsloë, a village in Sweden, where his own father had also been a clergyman. On his mother's side Winslow was the grand-nephew of the Swedish anatomist Sténon. Thus his family professions inclined him both to theology and to medicine, and this situation was resolved in a very strange way. Winslow began by studying theology. He had as a friend a medical student, and the two lads talked to each other about their respective studies to such effect that each developed a strong leaning towards the profession that was not supposed to be his. So firm was their purpose that they succeeded in winning over their parents, and Winslow transferred to the study of medicine while his friend changed to theology. Winslow was fortunate in gaining the support of two friends of his family. After he had studied for a year under Brirochius, he decided to work in the different universities of Europe. His sponsors undertook to help pay his expenses, and even obtained for him a pension from the King of Denmark. Winslow's first stay was in Holland, where for a year he was the pupil of Ruysch, who was well known for his techniques of preservation of cadavers. On this count Fontenelle said of him: "Mr. Ruysch's mummies to a certain extent prolonged life, whilst those of ancient Egypt merely prolonged death." Winslow then proceeded to Paris, where he intended to stay a short time only; actually he remained there for the rest of his life. Duverney, who was professor of anatomy at the King's Garden[2] for fifty years, was then at the height of his fame; he quickly took an interest in Winslow.

The story of the young Dane's conversion to the Roman Catholic faith is of some interest. Some time after his arrival in Paris, being anxious to see the Court of Louis XIV, he went with two Lutheran friends to Versailles; they even went into the chapel, and saw the King attending Mass with his court. Winslow was profoundly impressed, but remained sufficiently master of himself not to remain for the Canon of the Mass, which was against his Lutheran principles. The next significant event was a debate which he arranged with one of his compatriots; Winslow had decided that they should thrash out the most controversial points of the Roman Catholic and Protestant religions; he was to attack the Protestant religion, and his opponent to defend it. Winslow threw himself wholeheartedly into the collecting of his material, read the works of Bossuet and marshalled his arguments so successfully that he convinced not only his opponent, but also his own conscience. Because of the need to resolve the painful conflict between his Lutheran background and his doubts, Winslow sought help from Bossuet himself. The bishop thus found himself faced with the grand-nephew of Sténon, the Swedish anatomist, whom he had converted to Roman Catholicism forty years previously. In 1699 the Danish student (who was then thirty years old) renounced his Protestant religion and was baptised by the Bishop of Meaux, taking the names of his catechist, Jacques Bénigne.

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[1] *Presse méd.*, November 16, 1955.

[2] *Le Jardin du Roy*, reestablished by Louis XIV in 1671; see M. J. AUSTRALIA, December 4, 1954.

As a consequence of his conversion, Winslow received no further financial help either from his family or from the King of Denmark. He was in an even more critical situation since he had not definitely chosen his profession; of the alternatives, medicine and theology, he chose the former. The University of Florence, on the strength of the reputation of his grand-uncle Sténon, offered him a chair; he consulted Bossuet, who advised him to stay in France and to apply to the Faculty of Medicine of Paris. As he was unable to meet the expenses involved in taking the doctorate in medicine, Winslow requested that payment might be deferred. This was allowed; and twelve years later, when he offered to pay what was owing, the Faculty refused to take any money, stating that adequate payment had been made "by the glory that M. Winslow's courses brought upon its schools".

On Duverney's recommendation, in 1707 Winslow was admitted to the Academy of Sciences. Duverney also chose Winslow to take his place when his health no longer permitted him regularly to give his courses in anatomy at the King's Garden; but when Duverney retired, the chair was given to Hunault. A contemporary gave the following comparison of Winslow and Hunault:

> M. Hunault astonished his hearers by the magnificence and even the splendour of his oratory; M. Winslow's attraction lay in the clarity and precision of his. The former combined with learning the outward appearances and the ability to turn it to account; the latter, having only the qualities essential to his art, confined himself to a severe exactness of facts.

It was not until 1743 that Winslow acceded to the chair of anatomy at the King's Garden.

The results of Winslow's principal researches were presented to the Academy of Sciences. In 1711 he wrote a note on the heart, in which he showed that it was composed of three "muscles", and that the two ventricles were two separate vessels, each formed by a muscle and covered over by a third common to both. In 1715 he presented a new account of the position of the heart, and a note on the respective positions of the cardia and the pylorus. In 1720 he devoted an important study to the action of muscles. He showed the synchronization of the movements of antagonistic muscle groups. In 1723 a dancer won considerable success in Paris by grasping a rope between his scapulæ sufficiently strongly for him to be pulled along by it. Winslow used this event as the basis for a study of the shoulder muscles, recorded in three papers which he later presented to the Academy of Sciences. His most important work, *L'Exposition anatomique de la structure du corps humain*, was published in 1732, and was quickly translated into several languages. The book is more a plain statement of the author's observations and dissections than a résumé of his lectures. Osteology holds an important place, but the best part of the work is that devoted to mycology, especially the muscles of the larynx. The relations between the nerves and the peripheral vessels are shown *in situ*. Winslow originated the term "sympathetic nervous system", which replaced the term "intercostal nerves". He was the first to show the associating neurogenic fibres of the sympathetic and parasympathetic nervous systems. Winslow also showed that the *foramen epiploicum* (foramen of Winslow) was a normal anatomical arrangement, and not a tear produced during dissection, as had been previously held.

Two of Winslow's studies published after his famous book are of particular interest. In the first he analysed the movements of the hands, and this led him to postulate the existence of transverse connexions between bilaterally corresponding sections (*parties symétriques*) of the nervous system. In the second paper he drew attention to the dangers of corsets stiffened with whalebone and of tight-lacing. Finally Winslow wrote a study in two volumes on methods of making certain that a dead person was really dead. This work is thought to have been induced by personal experience, Winslow "having twice been thought dead and almost buried alive during his youth". As a consequence he gave it as his opinion that life could be present until putrefaction set in. In 1745 Winslow played the role of peacemaker. Bitter rivalry had long existed between the King's Garden and the Faculty of Medicine of Paris, the former having the advantage. In that year the Faculty of Medicine constructed a new teaching building, and invited Winslow (aged seventy-six years) to open it. In accordance with custom, notices were put up in the following terms:

> God willing, Jacques Bénigne Winslow, doctor and teacher, will give, to inaugurate the new medical school building, a course in French, open to the public, and will himself carry out the dissection and demonstration of the parts of the human body on a male cadaver, as he previously did in the old building in the *rue de la Bûcherie* opposite the little bridge of the *Hôtel-Dieu*. Those attending are forbidden to carry sticks and swords.

It was feared that there might be a riot, and the Dean thought it wise to separate the seats provided for the physicians from those for surgeons and students. However, the peace remained unruptured. Winslow continued to lecture, especially at the King's Garden, until 1758, when he was eighty-nine years old. He died at Paris in 1760, practically penniless, because he would not ask for a pension, which he had well deserved, and which the King would have been willing to grant him. To Winslow the words of Horace can be well applied (although he may never have thought of himself in that way): *"Exegi monumentum aere perennius."*

CORONARY HEART DISEASE AND OPERATIVE RISK.

IN the past there has perhaps been some undue pessimism when the future of the man with coronary disease was contemplated. Professors with angina continue to outlive their students and blood flows still through the diseased coronary arteries of persons in high places. In the minds of the surgeon and the anæsthetist there is considerable trepidation before an operation upon the patient who has either a history or signs of coronary artery disease. Even minor techniques may be postponed in the hope that Nature may indefinitely delay the evil day. However, patients with such a cardiac history appear to survive emergency surgical experiences remarkably well. It may now be the time to allay the bogy of such patients lying inert, stiff and beyond recall under the horrified hands of anæsthetist or surgeon. B. Etsten and S. Proger[1] publish their conclusions after operation upon a very large series of patients, many of whom had the history or findings of coronary artery disease. All the operations took place at the New England Center Hospital. The patients were divided into three groups for the purpose of comparison. In the first group were 517 patients who had a history either of coronary ischæmia or of old or recent infarction of the myocardium with electrocardiographic changes. In the second group were 1107 patients who had no history of coronary disease but in whom the electrocardiogram demonstrated such abnormalities as ST segment changes, inverted T waves, bundle branch block or auriculoventricular block. In the third group were 4154 patients with no history or signs of heart disease. The average age of patients in the third group was ten years lower than that of the patients in the other two groups. The total death rate for each group was corrected for this age difference. Of the 517 patients in Group I, 15 patients died after operation. Of these deaths six were cardiac in origin, five being attributable to acute post-operative myocardial infarction and the other being the result of acute pulmonary œdema. Two of the deaths in this group were from the 11 patients who, of necessity, were operated upon during the acute phase of the myocardial infarction. Of the 506 patients with chronic coronary artery disease there were thus only four post-operative deaths due to cardiac causes. In the second group of 1107 patients there were seven post-operative fatalities, four being due to myocardial infarction and three to acute pulmonary œdema. In the third normal group there were four post-operative deaths all due to acute pulmonary œdema. The incidence of deaths due to pulmonary embolism did not differ in the

[1] *J.A.M.A.*, October 29, 1955.

three groups. Myocardial infarction, without death, occurred in five other patients from the first group. The post-operative cardiac death rate in the chronic coronary disease patients was 0·8%, compared with 0·1% in those patients without pre-operative evidence of heart disease. There was no significant statistical difference in the effect of the duration of operation or of the choice of anæsthetic agent. Cyclopropane was not found to produce cardiac arrhythmias. The actual operations performed were of every type normally encountered in a busy hospital. In each of the groups about 60% of the operations were major in extent. Etsten and Proger found that there was no significant statistical effect of particular operations, such as cholecystectomy, on survival in patients with and without heart disease. They point out that a hazard, which presents in the general anæsthesia of the coronary patient, is circulatory depression which may occur during deep general or high spinal anæsthesia. Respiratory depression may also follow deep anæsthesia, and accumulating carbon dioxide would then predispose to cardiac arrhythmia and post-operative hypotension. Straining or coughing during the induction of anæsthesia is an additional risk in the patient with disease of the coronary valves, hypoxia may result and a final precipitating cause of coronary infarction would be shock. To combat these additional risks the authors suggest that anæsthetic induction should be as smooth and as rapid as possible, and that anæsthesia should be kept at light levels and respiration accurately controlled. They further suggest, as coronary atherosclerosis is so common in the middle-aged patient, that in all operations on patients in the older age groups precautions must minimize the possibility of post-operative myocardial infarction or coronary thrombosis. It would appear that under good medical conditions the patient who has survived for some time after the onset of coronary artery disease may, without undue fear, be submitted to any surgical technique which would, under normal circumstances, be considered to be worth while.

THE CHEMICAL CONSTITUTION OF INSULIN.

THE proteins have an essential part to play in living matter as the principal substances in cell structure, in framework structures, as enzymes and hormones and in many other ways. They are of immense complexity of structure, and until recently the more we learned about them the more impossible it seemed to determine their chemical constitution. As a knowledge of chemical constitution was necessary before synthesis could be achieved *in vitro*, the laboratory preparation of proteins seemed to be unattainable. We are still probably a long way from the synthesis of any known protein, but it no longer seems impossible. The synthesis last year of the octapeptides, oxytocin and vasopressin, by du Vigneaud, described in this journal on July 2, 1955, was an important step along this road. The determination of the chemical constitution of insulin by Sanger *et alii* is another important advance. Sanger and his associates have published a number of papers from 1945 onwards in which are described the various steps in the elucidation of the constitution of insulin. In a paper recently published[1] by A. P. Ryle, F. Sanger, L. F. Smith and R. Kitai the final step is announced of the determination of the situation of the disulphide bonds in insulin. Towards the end of this paper is the simple statement: "The results are summarized in Table 10 which gives the complete structure of insulin." The insulin referred to is from the ox pancreas, and in a following paper H. Brown, F. Sanger and R. Kitai give the constitution of pig and sheep insulins. The actual formulæ are too long for reproduction here, but the following may give some idea of the structure and also of the immensity of the task of working it out.

By mild oxidation of the disulphide linkages the molecule of insulin can be broken into two chains, one called fraction A or the glycine chain, the other fraction B or the phenylalanine chain. There are 21 amino acid residues

joined by peptide linkages and consisting of ten different amino acids in fraction A and 30 amino acid residues consisting of 15 different amino acids in fraction B. Altogether there are sixteen different amino acids in the molecule of insulin. In the whole molecule the two fractions are united by two disulphide linkages. There is another disulphide linkage bridging over four amino acids in fraction A. This is a particularly interesting one, for in the cattle insulin the linkage passes over cystine, alanine, serine and valine; in sheep insulin cystine, alanine, glycine and valine; and in pig insulin cystine, threonine, serine and isoleucine. The rest of the molecule is the same in all three insulins. In spite of these differences the three insulins appear to act in the same way in the human body and in immunological behaviour. They have also the same crytalline form. It is probably of importance that the same structure occurs in vasopressin and in oxytocin and may have physical or biological significance.

One of the difficulties in the later stages of these researches had to do with the molecular weight of insulin. Earlier investigations had made it probable that the molecular weight was 36,000 or 48,000, and if either of these was correct the difficulties of the work would have been enormously increased. More recent work has indicated that the molecular weight is about 6000. There seems no reason to doubt that the work of Sanger *et alii* is definitely correct and that the molecular weight of cattle insulin is 5734, so that insulin is a relatively simple protein. The amino acids present in cattle, sheep and pig insulins had been determined by several workers and fairly accurate estimations made of the relative proportions of the different amino acids. The outstanding achievement of Sanger *et alii* consists in the determination of the exact sequence of the amino acids and the positions of the disulphide linkages. The work was carried out in a brilliant manner; when difficulties were encountered, new and remarkably efficient methods were devised to meet the particular problems and thoroughly tested. The method of ionophoresis was very considerably developed to separate the peptides resulting from enzymic breakdown of the protein.

The octapeptides oxytocin and vasopressin have been synthesized by du Vigneaud, but these are relatively simple substances as compared with insulin. Much has yet to be learned about the insulin molecule, such as the spatial arrangement of the amino acids, before synthesis can be attempted, but it will be done. The methods developed by Sanger *et alii* will undoubtedly be used by others for the examination of other proteins.

MEASLES AND BACTERIAL INFECTION.

THE question of the routine administration of chemotherapy in children with measles is one which has vexed general practitioners and pædiatricians for some time. Acute suppurative *otitis media* and severe infections of the respiratory tract still remain the serious complications of what is otherwise a relatively mild disease. Measles appears to be losing its virulence, and epidemics vary considerably in character from one outbreak to the next. This difference in character considerably clouds the chemotherapeutic issue. During the last year correspondence in the *British Medical Journal* waged hot and strong, and the contestants both for and against therapy waged battle royally. At times it is hard to apply the findings of others to one's own experience, but that is the chief characteristic of measles, its divergence from the expected. It comes as a surprise to realize that the controversy has been raging for fifteen years. As far back as 1942 H. Gibel and A. M. Litvack[1] found that sulphathiazole used routinely had no effect either upon the disease itself or upon the usual complications owing to secondary infection. They concluded that the routine use in all cases of measles was not warranted. Some time later A. F. Coburn[2] reported the results obtained in the prophylaxis of streptococcal infections by the routine daily administration of sulphadiazine

[1] *Biochem. J.*, August, 1955.

[1] *J. Pediat.*, September, 1942.
[2] *J.A.M.A.*, September 9, 1944.

to 30,000 servicemen. The observations suggested that one gramme of sulphadiazine daily was adequate to check a well-advanced streptococcal infection epidemic and to protect 85% of susceptible persons from infection by bacterial respiratory pathogens. More recently, in answer to a question in the *British Medical Journal*,[1] it was stated that the period of danger of secondary infection was comparatively short and that prophylaxis should be confined to severely ill patients between the ages of six months and two years. It was suggested that routine prophylaxis was more strongly indicated for children already in hospital, who were more likely to be exposed to secondary bacterial infection. The view was expressed that, because of their toxicity and their relative inefficiency, the prophylactic use of sulphonamides should be replaced by that of penicillin. The author further indicated that penicillin should be administered at the first sign of a developing *otitis media*. In some quarters this will be questioned. Many Australian otologists will not agree with it. The point is that once administration of penicillin in *otitis media* is begun it must be continued, and there is no doubt that by this means the incision of many tympanic membranes will be avoided.

G. I. Watson,[2] in reporting a series of 142 cases of measles in a country practice, stated that 90 of the children were prophylactically treated with sulphadiazine. Watson found that when no prophylaxis was used 80% of the children, who had previously shown signs of chronic bacterial infection, developed secondary complications, while only 11% of the previously healthy children did so. The use of sulphadiazine reversed this trend, and while reducing all complications, did so particularly in children who were previously infected with bacteria. Watson also noticed that measles, if anything, was less severe in those children under three years of age. He concluded that the sulphadiazine acted not so much as a prophylactic against future disease, but as a curative of existing chronic disease.

The most recent study of the value of prophylaxis for patients with measles has been made by L. Weinstein.[3] Extensive observations were made upon 428 patients admitted to hospital with measles. Secondary bacterial infection was recognized to exist only if, in addition to isolation of the pathogens, there were signs that the infection included a change in the level of the neutrophile cells. It was found that 24.6% of the patients already had evidence of bacterial infection on admission to hospital. Of the patients who had received antibacterial drugs prior to admission, 30.4% were already infected by bacteria, as opposed to 14.9% of infection in those previously untreated. Of the 350 patients whose illness was uncomplicated on admission, 16 became secondarily infected in hospital, predominantly with pneumonia and suppurative *otitis media*. This low rate of secondary infection does not bear out the original thought that cross-infection is more likely in the hospital. Of the 94 patients who either entered with, or subsequently developed, secondary bacterial infection, 11.6% also developed bacterial superinfection during antibiotic treatment, thus demonstrating the abnormal uncontrollable increased risk of bacterial infection in the measles patient. Superinfection occurred especially in children with a bacterial pneumonia. Weinstein concludes that the use of antibacterial drugs in patients with measles not only fails in the prophylaxis, but is actually accompanied by an increased risk of bacterial infection. He suggests that these drugs should be used only when there are definite signs of secondary bacterial infection in the measles patient.

To be sure that chemoprophylaxis is useful, useless or dangerous a controlled survey of a very large number of cases of measles from several separate outbreaks must be studied. Such a survey is at present being undertaken by the College of General Practitioners in the United Kingdom. The outcome of this inquiry will be awaited with great interest. In the meantime the results obtained in these other smaller series do not indicate that chemo-

therapy has a place in the routine prophylaxis of bacterial secondary infection in cases of measles.

THE TREATMENT OF GASTRIC ULCERATION.

Two papers recently published indicate the valuable research which may be done by means of planned experiments without necessarily employing complicated or highly specialized techniques. They are essentially studies in the natural history of disease and in the possibility of modifying it by routine methods of treatment in routine hospital practice. R. Doll, P. Friedlander and F. Pygott[1] report the effects of a "standard" hospital ulcer diet on patients with gastric ulcers in comparison with the effects on a comparable group treated with the normal hospital diet less fried foods. Although there was evidence to suggest a greater weight gain and perhaps greater radiological healing of the ulcers in those treated with the normal diet, relief of pain was more satisfactory in those treated with the ulcer diet. The latter group, it should be mentioned, had two-hourly feeds, so that the normal diet patients, despite between-meal snacks, had two feeds per day less. In an out-patient investigation, which the authors point out is an assessment not of diet so much as of advice about diet, the results were little different. No difference was noted between the two groups in regard to symptomatology at the end of a year, although slightly more of the patients on the ulcer diet required readmission for pain or hæmorrhage. Again the evidence suggested that gastric ulcers healed a little more readily in patients given the normal diet, in this experiment a wholly normal diet. The cautious conclusion to be drawn from all this is not that diet is unrelated to the pathogenesis of gastric ulcer, but that "specifically harmful or curative factors—if they exist" in the diet have not yet been identified, and that nothing is gained by limiting the hospital diet to bland foods only. It could be suggested on these findings that patients with gastric ulcer fared better on the normal diet because they had not eaten a normal one previously.

The second paper, also by R. Doll[2] (with A. V. Price, F. Pygott and P. H. Sanderson) relates to the role of a continuous intragastric milk drip in the management of uncomplicated gastric ulcers, on which H. W. Garlick[3] reported favourably in this journal some years ago. The experimental design in this instance was somewhat more complex because other methods of treatment were investigated simultaneously, but the results are reasonably clear. Relative to the controls, no significant improvement was found in the healing of ulcers by using a drip of milk or of milk alkalinized with sodium bicarbonate (20 to 40 grammes daily), but the method had a probable advantage in the relief of pain and in producing weight gain. No serious side effects were noted. It would seem therefore that while the gastric secretions may be related to the pain of gastric ulcer, they are not related to the perpetuation of ulceration in the stomach.

Readers interested in the relatively simple design of these experiments are referred to the original papers. Both investigations are of a type which requires little more than ordinary hospital facilities, a keen observer, and initial advice from a statistician, to ensure useful results.

A. M. Dawson[3] has treated two comparable groups of 50 patients suffering from gastro-intestinal hæmorrhage by means of a continuous intragastric drip of milk, in addition to the usual medical measures. No significant difference was noted between the groups in mortality or in the incidence of further hæmorrhage or emergency operation. Aspiration of blood through the Ryle's tube used was also not significantly more frequent in the patients treated with the drip. It is of interest that this sign was found to be of no value in giving warning of an impending massive bleeding; this appears to occur so rapidly that the usual signs of increasing pulse rate and falling blood pressure develop at the same time.

[1] *Brit. M. J.*, "Any Questions", May 2, 1953.
[2] *Brit. M. J.*, January 1, 1955.
[3] *New England J. Med.*, October 20, 1955.

[1] *Lancet*, January 7, 1956.
[2] *Lancet*, January 14, 1956.
[3] THE MEDICAL JOURNAL OF AUSTRALIA, 1949, 2: 452.

Abstracts from Medical Literature.

OBSTETRICS AND GYNÆCOLOGY.

Reducing the Risk of Total Abdominal Hysterectomy.

P. TERZIAN AND J. P. TIMPANE (*Am. J. Obst. & Gynec.*, April, 1955) report studies of groups of approximately 40 patients by five different types of vaginal preparation before total abdominal hysterectomy in an attempt to reduce post-operative morbidity. They state that the mortality rate following total abdominal hysterectomy nowadays is usually less than 2%, but the post-operative morbidity rate is still rather high and averages 35%. The criteria of morbidity used in the study are similar to those of the Joint Committee on Maternal Welfare—namely, an oral temperature of 100·4° F. or over, on any two consecutive days after the first post-operative day. The post-operative morbidity rate of the series of 483 consecutive total abdominal hysterectomies was 36%. The outstanding cause of fever was pelvic infection. The only significant difference among the several groups of patients was the type of pre-operative vaginal preparation given. Certain constant and variable factors apply to each of the operations reported, and these factors are briefly discussed. Part of the risk of hysterectomy is intrinsic in the patient, owing to such factors as obesity, metabolic disease and the type of pelvic pathological condition. A greater part of the risk of hysterectomy is extrinsic, depending on surgical technique and pre-operative and post-operative care. Patients were divided into five groups according to the type of vaginal preparation given before operation : group 1 had a pre-operative vaginal tampon soaked in " Sulfamylon " ; group 2 had a dry cotton vaginal tampon inserted the afternoon before operation ; group 3 were disinfected in the operating theatre with tincture of " Zephiran " ; group 4 had a penicillin-streptomycin suppository inserted in the posterior fornix the afternoon before operation ; group 5 had no pelvic examination in hospital and no pre-operative vaginal preparation. The pelvic morbidity rate was in the order of 17% to 24% in groups 1, 2, 3 and 5. The best results were obtained with the use of a penicillin-streptomycin suppository inserted into the vagina before operation, the morbidity rate being 6%.

The Present Status of Human Male Infertility.

J. MacLEOD (*Am. J. Obst. & Gynec.*, June, 1955) presents an analysis of the present-day accepted criteria of male fertility. In terms of the sperm count this is much wider than one might suppose. Sixty million sperms per millilitre is now considered to be too high a figure. In a large series, 29% of fertile men had counts below this figure, 17% had sperm counts below forty millions, and 5% had counts below twenty millions. The author considers that the lower limit could be placed at twenty millions.

There is a much closer relationship between the quality of sperm motility and the fertility potential. The motile activity of spermatozoa is of more significance than the sperm count, and may compensate for other seminal deficiencies. Impaired fertility is most often found when the proportion of active cells drops below 40%, and it is at this level that a sharp difference between fertile and infertile population is found. Morphology is of considerable importance, and the author suggests that poor fertility due to abnormal morphology may be caused by the relatively poor motility shown by abnormal cells. No positive instance has been presented which links abnormal sperm morphology with abnormalities of pregnancy. Although a sperm count does increase with prolongation of continence (up to double after ten days), there is also decreasing motility ; so that what is gained in the sperm count may be lost from the increasing number of inert cells. The fertility potential is not increased after a period of two or three days' continence. A higher rather than a lower rate of intercourse is advised. Intercourse three times a week would maintain viable sperms in the female reproductive tract through the major portion of the cycle.

Female Genital Tuberculosis.

B. P. ZUMMO, H. SERED AND H. FALLS (*Am. J. Obst. & Gynec.*, July, 1955) present a study in the diagnosis and prognosis of 64 patients with genitoperitoneal tuberculosis, from the Cook County Hospital. It is generally agreed that 5% or less of all disease of the Fallopian tubes is due to tuberculosis and that 1% to 6% of all sterility problems are due to the same disease. The diagnosis of tuberculosis may be most difficult because of the manner in which it masquerades as other gynæcological abnormalities. The authors found that 30% of cases of tuberculosis of the female genital tract were not diagnosed until operation had been performed. The most common conditions for which the disease was mistaken were partial or complete intestinal obstruction, chronic pelvic inflammatory disease, recurrent appendicitis and fibromyomata of the uterus with pelvic inflammatory disease. Twenty-five patients (27·5%) had pulmonary involvement and 10 (6·4%) had gastro-intestinal abnormalities suggestive of tuberculosis. Endometrial and cervical biopsy examinations are useful procedures (34 gave positive results in 64 patients with this tuberculosis) and are complementary to bacteriological studies. Hæmatological examination revealed a relative or absolute leucopenia, and experience has shown that at least 50% of patients with active tuberculous infection will have an abnormally low hæmoglobin level. Of 28 patients treated before the advent of anti-tuberculosis, chemotherapeutic and antibiotic drugs (1949), 18 died, four lived and six were untraced over a period of three years. The combination of streptomycin with PAS and isoniazid has dramatically changed the prognosis. Silent lesions in the endometrium and oviducts respond promptly, and pregnancies are now known to have occurred provided that the pathological changes have not

been too advanced. Many patients have been spared the ordeal of surgical treatment. However, adnexal masses, abscesses, prominent thickenings, infiltrations and ascites, in many instances still require surgical treatment. Operative and post-operative hazards are reduced.

Infectious Hepatitis in Pregnancy.

J. S. LANG, H. BOYSEN AND F. O. PRIEST (*Am. J. Obst. & Gynec.*, August, 1955) review the relevant literature and present nine cases of pregnancy complicated by infectious hepatitis. The series included one case in the first trimester, three cases in the second trimester, and five cases in the third trimester. The length of the active phase of the disease was between two and four weeks. Those patients in the first and those in the second trimester of pregnancy recovered and had normal deliveries of normal babies. In the third trimester, the acute cases were the most severe. Two of the five patients died owing to hepatic insufficiency. The treatment of infectious hepatitis occurring during pregnancy is mainly prophylactic with general supportive measures. Abdominal delivery is contraindicated, and preparations should be made, if labour does ensue, for the control of hæmorrhage. Because of the few successful cases recorded in the literature the use of " Aureomycin " would seem to be indicated.

Carcinoma of the Breast in the Pregnant and Nursing Patient.

T. T. WHITE (*Am. J. Obst. & Gynec.*, June, 1955) reviews 1375 cases which have been reported in world literature of carcinoma of the breast occurring during pregnancy. The disease complicates about three out of every 10,000 pregnancies, whilst pregnancy itself occurs in about three out of every 100 patients with carcinoma of the breast. The gross five-year survival rate (59%) and ten-year survival rate (47%) of patients who became pregnant after treatment for carcinoma of the breast are comparable to the gross survival rates when pregnancy did not occur. The ten-year survival rate of treated patients with coincidental carcinoma and pregnancy is 8·6%. Where no metastases are discovered 21·5% of the patients observed since 1941 have survived for ten years. Those patients with metastases have five to twenty times as poor a prognosis as those without. The average age of 751 patients was 35·8 years. For 521 patients the delay between examination by the doctor for tumour and treatment was 3·1 months. The prognosis for patients treated for the first time in the second and third trimesters appears less favourable than that for those treated either in the first trimester or during lactation. No benefit could be demonstrated from terminating the pregnancy of the patient with breast carcinoma complicated by pregnancy.

Acute Renal Failure and Concealed Accidental Hæmorrhage of Pregnancy.

G. M. BULL, A. M. JOEKES AND K. G. LOWE (*Lancet*, December 3, 1955) present a series of 22 cases of acute renal failure

following concealed accidental hæmorrhage in the last three months of pregnancy. Three of the patients each had one living child, but in no case was there either *placenta prævia* or rupture of the lower segment of the uterus. Seven of the patients had only mild signs of acute renal failure, with short-lived suppression of urine and with the maximum blood urea level between 25 and 82 milligrammes per 100 millilitres of blood. Eleven patients had signs of moderate or severe renal failure, with oliguria lasting for several days and with maximum blood urea levels of 120 to 450 milligrammes per 100 millilitres of blood. Kidney tissue damage in these patients was considered to be proportional to the oliguria and the rise of blood urea level, and in the more severe cases there would be acute tubular necrosis or incomplete cortical necrosis. All these patients made a satisfactory recovery with conservative treatment. Four of the patients died, and necropsy revealed that in each case there was gross cortical necrosis incompatible with recovery. In two cases death was accelerated by overloading with salt and water. One of the patients, who subsequently died, was treated with an artificial kidney. Death took place after six to forty-two days of oliguria, and the maximum blood urea level was from 213 to 485 milligrammes per 100 millilitres of blood. It was noted that the severity of kidney damage was not associated with age, parity, the amount of concealed or overt blood loss or the duration of time from the onset of bleeding to delivery of the uterine contents. In the patients who recovered, oliguria did not last for more than six days, with one exception, and this patient had a peak blood urea level of 450 milligrammes per 100 millilitres of blood. Biochemical tests revealed that the pattern of disturbed renal function in acute tubular necrosis owing to concealed accidental hæmorrhage was the same as that pattern due to other causes. The blood pressure, which might have been expected to fall owing to the hæmorrhage, in some cases was raised and maintained at hypertensive levels. This was probably due to generalized vasoconstriction, especially in the renal vascular bed. The authors suggest that renal sympathetic block may usefully be employed in order to combat the renal damage. The patients who died each had marked signs of the toxæmia of pregnancy. The Bull regime was not strictly adhered to in the treatment of the patients in the series. In mild cases there was no dietary restriction, though some were treated, as in acute nephritis, with an oral diet low in protein and high in calorific value, and with some approximate salt and water balance. When no diuresis had occurred within twenty-four to forty-eight hours, a strict diet control was instituted. The original tube feeding regime of fat-sugar emulsion was still used for some patients, but more attention was paid to the 500 millilitres of water replacement than to fat content. It is suggested that sodium-charged cation-exchange resin may be given either by the oral route or by enemata to prevent potassium intoxication. Many patients tolerate tube feeding poorly, and for them the authors favour the adminis-

tration of hypertonic glucose infusion by the venous route. Either the superior or the inferior *vena cava* may be used for this purpose in very severe cases of acute renal failure. The authors suggest that dialysis is sometimes useful, but that its routine use is controversial. Finally, they advise that the correction of acidosis and hyponatræmia may be desirable, but that calculated salt doses should be given with caution.

Fibrinogenopenia in Intrauterine Death.

J. A. PRITCHARD AND O. D. RATNOFF (*Surg., Gynec. & Obst.*, October, 1955) studied fibrinogen concentration in the plasma of 31 women three or more weeks after intrauterine death of fœtuses had occurred. Serial determinations of fibrinogen were made before, during and after labour and delivery. Hypofibrinogenæmia was considered to exist when the fibrinogen level fell below 150 milligrammes per 100 millilitres of plasma. In 23 of the women there was no critical depletion of circulating fibrinogen. In seven of these serial fibrinogen levels were estimated for several weeks after fœtal death, and in each case there was a progressive linear reduction in the fibrinogen level. Eight of the women studied developed dangerously low fibrinogen levels, and three of these already had hæmorrhagic manifestations. In three of the non-symptomatic patients, fall in fibrinogen plasma level continued until hysterotomy was performed. Twelve women from the series, in two of whom there was hypofibrinogenæmia, were studied for changes in plasma fibrinogen during labour. In none was there any change in the fibrinogen level. Of the eight women with hypofibrinogenæmia, three were Rh-positive reactors. In at least five of them the condition was not sudden, and in none was there evidence of any spontaneous correction of fibrinogen level before labour. All eight women had normal or raised fibrinogen levels in the plasma within forty-eight hours after delivery. There was no specific rise in plasma fibrinolytic activity. Liver function in those tested was normal. Amniotic fluid obtained from one of these patients before rupture of the membranes was not fibrinolytic, nor was it an activator of the fibrinolytic properties of normal plasma. In seven of the women there was no marked thrombocytopenia, though levels were below the expected normal. There was no marked hypoprothrombinæmia in five of these patients, but both apparent and true plasma prothrombic activity was rather depressed. In no patient was there found to be the moderate hyperprothrombinæmia usually discovered in the plasma of pregnant women. The authors suggest that in the management of these patients with hypofibrinogenæmia, the patient must report immediately there is any hæmorrhagic symptom, and further that in all cases weekly estimations of plasma fibrinogen must be made after the third week of intrauterine fœtal death. If the plasma fibrinogen level falls below 150 milligrammes per 100 millilitres of plasma, then the uterus must be emptied promptly. Data available suggest that vaginal labour will not increase the abnormality ;

but if induction fails, abdominal hysterotomy must be performed. There must always be four grammes of fibrinogen and several pints of compatible blood available before labour is induced, and it is safer to give three to four grammes of the fibrinogen by the intravenous route before the delivery, and certainly if bleeding occurs other than from the myometrium. Fibrinogen must always be given before abdominal hysterotomy is performed, and a dextrose or saline intravenous continuous drip apparatus should be set up and diluted " Pitocin " administered for several hours after operation. Should vaginal bleeding occur, the authors suggest that the circulating fibrinogen should be estimated by a clot observation test, and fibrinogen be administered by the intravenous route if indicated. They state that clotting time estimation gives no useful indication of the presence of hypofibrinogenæmia. If human fibrinogen is not available, whole blood may be used instead.

Sarcoidosis of the Uterus.

A. ALTCHEK, J. A. GAINES AND L. E. SILTZBACH (*Am. J. Obst. & Gynec.*, September, 1955) discuss sarcoidosis in general, and report a case of sarcoidosis of the uterus. They define sarcoidosis as a generalized, chronic disease of unknown ætiology, characterized by the presence of epithelioid-cell granulomata which usually involve lymph nodes, lungs, eyes and skin. They state that in histological studies sarcoidosis may be mistaken either for tuberculosis or for foreign body granulomata. The case reported is one of generalized sarcoidosis with asymptomatic uterine involvement. The patient was a multiparous Negro woman, aged thirty-nine years. She was submitted to the operation of combined vaginal hysterectomy and repairs for genital prolapse and an associated fibromyoma. Conspicuous granulomatous lesions were found scattered throughout the endometrium and myometrium. These consisted of epithelioid-cell tubercles, without caseation, but with associated giant cells. There had been a history of a chronic cough for ten years, and a diagnosis of sarcoidosis of the lungs had been made five years earlier. The lung condition had regressed and X-ray examination of the chest revealed no abnormality at the time of pelvic operation. The authors state that young adults are those most often affected and that regression of the disease may occur at one site while new foci develop at other sites. The result of the Nickerson-Kveim intracutaneous test, made with a sarcoid tissue suspension, has been found to be positive in 86% of affected subjects. It is thought that the condition does not influence fertility, pregnancy, labour or the health of the newborn infant. Conversely, pregnancy does not appear to influence the course of the disease. In contrast to pelvic tuberculosis there is no caseation in sarcoidosis, acid-fast bacilli are not found, and the uterine tubes do not become involved. Moreover, pelvic tuberculosis is usually symptomatic. Foreign-body granulomata usually follow infection, manipulation, surgical interference or the insertion of foreign bodies.

Clinico=Pathological Conferences.

A CONFERENCE AT SYDNEY HOSPITAL.

A CLINICO-PATHOLOGICAL CONFERENCE was held on August 16, 1955, at Sydney Hospital, the medical superintendent, DR. NORMAN H. ROSE, in the chair. The principal speakers were DR. H. M. WHYTE, Senior Clinical Research Fellow, and DR. F. C. COURTICE, Director of the Kanematsu Memorial Institute of Pathology, Sydney Hospital.

DR. NORMAN H. ROSE: The case today is to be discussed by Dr. Whyte, who is in charge of clinical research at Sydney Hospital. The presentation is rather unusual in that the commentator did see the patient during life, but he has no knowledge of what was found at the post-mortem examination. We have in the audience several people who have worked, or are working, on problems related to hypercholesterolæmia, which this patient had, so I think we can be assured of a high-class discussion.

Clinical History.

The following clinical history was presented:

The patient, a married woman, aged forty-five years, was referred to the medical out-patient department in June, 1952, because she was noticed to have an orange discoloration of the palmar creases of the hands and similar lesions on the right elbow. She complained of lassitude and increasing weight, and although she had previously disliked hot weather she now felt the cold severely. Examination revealed a dry skin, a puffy face and supraclavicular pads of fat. The blood pressure was 160 millimetres of mercury, systolic, and 80 millimetres, diastolic; there were extrasystoles every third beat, and the thyroid was palpable but not enlarged. Her serum was clear; the total serum cholesterol content was 550 milligrammes per 100 millilitres; the result of a glucose tolerance test was normal; the basal metabolic rate was −15%; and an electrocardiogram was normal with a low T wave in CF. ● An electrophoretogram showed a relative increase in the globulins, especially the α 2 and β. She was treated with thyroid extract, half a grain twice a day; her condition improved; the serum cholesterol level remained high. Later she developed intermittent claudication and was treated with "Priscol".

In May, 1953, she was admitted to hospital for investigation. Heparin given by injection or by addition to her serum in vitro did not alter the serum cholesterol level. The only change after injection of heparin was an alteration of the lipoprotein pattern in the electrophoretogram. The dose of thyroid extract was increased to two grains three times a day; its administration was suspended because of toxic symptoms. It was later recommenced at a smaller dosage.

In September, 1954, she was admitted to hospital complaining of sudden onset of severe retrosternal pain, which radiated between the shoulder blades, down the arms and up the back of the neck; the pain lasted twelve hours. She vomited before and during the attack, which was associated with cold sweats. Her pulse was regular at a rate of 84 per minute, and the blood pressure was 140 millimetres of mercury, systolic, and 100 millimetres, diastolic. There was smoothing of the contours of the palmar aspects of both hands, and yellow pigmentation of the soles of the feet. Yellow deposits were present in the conjunctival fornices and in the upper lip. An X-ray film of the chest was normal. Electrocardiograms were taken on four different occasions, and each time were reported on by a different physician: two found evidence of a posterior infarct, one thought the tracing supported this diagnosis but the "evidence is not unequivocal", and the other's report was "within normal limits".

Results of other investigations were: hæmoglobin value, 10·8 grammes per centum; total leucocyte count, 8500 per cubic millimetre, with a normal differential count; blood sedimentation rate, 20 millimetres in one hour (Westergren). The serum cholesterol level was still much raised. The patient was afebrile during her stay in hospital.

During 1954 she had two further "heart attacks", but she was not admitted to hospital. Examination revealed that the left femoral pulse was less palpable than the right, and the popliteal and tibial pulses were impalpable. A Graham's test showed the gall-bladder to be normal. After a course of ethinyl œstradiol the yellow palmar discoloration decreased.

About the end of June, 1955, she complained of many bouts of chest pain, usually brought on by exertion, but sometimes occurring at rest; the pain radiated as before and would sometimes last as long as ten minutes. On July 6 she suffered six attacks of chest pain. In the early hours of the next morning she collapsed, became unconscious and died after twenty-five minutes of stertorous breathing.

Special tests gave the following results: (i) Serum cholesterol content: total varied from 730 to 222 milligrammes per centum, ester from 676 to 184 milligrammes per centum. (ii) Blood urea nitrogen content, 10 milligrammes per centum; blood creatinine content, one milligramme per centum. (iii) Skull and long bones X-ray appearances: no abnormality detected.

Clinical Discussion.

DR. H. M. WHYTE: I knew this patient and liked her. She was a pleasant, uncomplaining person, of average build, with a rather muddy coloured skin and brown hair as though lightly tanned. She was not obese, but neither was she skinny. She was very alert. Her skin was, perhaps, a little dry and rough, but it was not scaling; her hair was dry, but it kept its natural curl and was not falling. In fact, from a distance, there was nothing remarkable about her appearance. But in the course of walking two or three hundred yards she would have to stop several times to ease the pain which exertion brought to her calves. And during the last year of her life she experienced several agonizing attacks of pain in her chest and back, and it was following a succession of these that she died.

I think that she died of a coronary occlusion, and that her previous attacks were due to coronary artery disease. There is no denying that she had intermittent claudication, and this, too, was due to arterial disease. The underlying cause, or at least a partial explanation of the cause, was to be found on inspection of her palms: for there she had xanthomatosis. Indeed a good deal of her history and of her future could be read in her palms. Some physicians who have studied this disease intensively would claim, though they deny the gift of second sight, that they could see in her palms the beginnings of the trouble, dating back to 1906. It was in that year, in Scotland, a land of Celtic mysteries, that two genes came together by chance to form a malicious partnership, which then wrought havoc in its human host for forty-eight years, before finally killing her in Australia this year. This disease from which I believe she suffered is essential hypercholesteræmia, or if you like, essential xanthomatosis of the hypercholesteræmic type or hypercholesteræmic familial xanthomatosis. At any rate, it is a disease in which the serum cholesterol level is abnormally high for no apparent reason, and xanthomatosis and cardiovascular lesions are common, and which frequently runs in families. It is a disease which is said to be not so rare that it might not claim among its victims one or two of the present audience.

The patient presented with xanthomatosis. As these slides show, she had yellow discoloration of the creases of her palms, and on the backs of her elbows there were areas which were rough with small yellow nodules as though finely grated carrot had been incorporated into the skin. She also developed small deposits on her eyes and on her lip and a nodule under the skin of one finger. The creases of her soles were yellow, too. Her lesions were not as gross as the tuberous xanthomata shown in the next picture. Nor did she have the conspicuous plaques close to the eyes, called xanthelasma, which commonly occur in this condition. Nor did she have lesions scattered over the axillæ, bends of the elbows and knees, sites which are characteristic of xanthoma disseminatum. Nor did she have eruptive xanthomatosis, fluctuating eruptions of small raised nodules usually the size of a pin's head and surrounded initially by an inflammatory halo, scattered over the back and buottocks and other areas and associated with hyperlipæmia—that is, creamy blood. Here is a sample of hyperlipæmic serum compared with clear normal serum.

Xanthomatous deposits, however they are distributed and whatever shape they take, contain fatty material and indicate some disturbance in the metabolism of fat. Examined in this way there are three types of xanthomatous conditions. The first is associated with milky serum—hyperlipæmia—where the evidence of an abnormality in the handling of fat is obvious to the naked eye. Hyperlipæmia may occur normally for a short time after a fatty meal. Persistent hyperlipæmia, sufficiently protracted to cause xanthomatosis, occurs sometimes as a secondary manifestation of uncontrolled diabetes mellitus, chronic pancreatitis, nephrotic disorders and, rarely, other diseases, and it occurs sometimes as a primary condition of unknown ætiology which is often familial. The other two type of xanthomatosis are associated with clear serum. In one of these, serum analysis provides evidence of the abnormal lipoid metabolism

in revealing an elevated serum cholesterol content—that is, the hypercholesteræmic type. Again, the abnormality may be secondary to some condition such as biliary cirrhosis or myxœdema, or it may be of the essential or idiopathic variety, in which no underlying cause has been found. The third type of xanthomatosis embraces those rare examples associated with clear serum and a normal serum cholesterol content which appear occasionally in the Hand-Schüller-Christian syndrome and allied lipoid disorders.

The patient we are discussing today had clear serum and a high serum cholesterol content. I think it would be helpful if at this stage Dr. Courtice would tell us something of the physiology of lipoid metabolism, the way in which fats are normally handled by the body.

DR. F. C. COURTICE: Firstly, let me tell you very briefly how lipids exist in the plasma. The plasma lipids comprise neutral fat, which is mainly in the form of triglycerides of palmitic, oleic and stearic acids; phospholipids, which are almost entirely lecithins; and cholesterol, which may be either free or esterified with fatty acids. Fats and cholesterol are water-insoluble, so do not exist as such in the plasma. Neutral fat is present as chylomicrons—small particles of fat which contain a little cholesterol and which are stabilized by a film of protein and phospholipid. These particles vary in size up to 1μ in diameter and can be seen under the dark-ground microscope. In the post-absorptive or fasting state they are few in number, but after a fat meal they are numerous. Practically all the ingested fat absorbed from the gut enters the blood-stream through the thoracic duct in the form of chylomicrons, which make the lymph and plasma appear milky.

There is, as we have seen, some cholesterol and phospholipid associated with the neutral fat in the structure of the chylomicrons. Most of the plasma cholesterol and phospholipid, however, is bound to protein as lipoprotein and does not vary appreciably during the course of a day with the absorption of fat from the alimentary tract. Electrophoresis shows that in man these lipids are associated with the α and β globulins to form α and β lipoproteins. These molecules are larger than the normal proteins, but, of course, are very much smaller than the chylomicrons. In composition they consist largely of lipid: lipoprotein is about 75% lipid and 25% protein.

Plasma lipids, therefore, consist of chylomicrons, which vary throughout the day with alimentary absorption, and lipoproteins, which in any individual do not vary appreciably with alimentary lipæmia.

In the post-absorptive state the plasma lipids are fairly constant in any one individual, but vary from individual to individual. In an understanding of lipid in disease we should ask ourselves: "What controls the levels in the plasma in the normal animal?"

As this patient had hypercholesteræmia, let us consider only cholesterol.

There are two equilibria which we should consider. The first is the balance between plasma and tissue cholesterol. There is a rapid exchange of cholesterol between the plasma and the tissues, especially the liver. This has been shown by injecting labelled cholesterol intravenously. But what controls this exchange we do not know. The thyroid gland no doubt plays a part, for depression of the thyroid activity raises the plasma cholesterol level, whereas over-activity lowers the plasma cholesterol level. There is some evidence that in such circumstances the tissue cholesterol does not alter appreciably, so the thyroid may have a regulating effect on the level of plasma cholesterol without altering the total cholesterol content of the body.

The second equilibrium is the balance between the formation and destruction of cholesterol. I think this is what Dr. Whyte has to consider in this patient. It is clear that in a steady state there must be a balance between the absorption and synthesis of cholesterol on the one hand and its excretion and destruction on the other.

The amount of cholesterol absorbed depends on the cholesterol content of the foodstuffs eaten and also on the fat content of the diet. Cholesterol is readily absorbed through the chyle provided fat and bile are present. When a fat-free diet is being taken, ingested cholesterol (in rats at least) may be recovered in the fæces. Cholesterol can also be synthesized in almost all tissues, but the liver is the most active. It can be readily synthesized from the simple substance acetate; so all three foodstuffs, carbohydrate, fat and protein, may be a source of cholesterol. The rate of synthesis depends on the amount of dietary cholesterol. Normally in man cholesterol synthesis is greater per day than dietary cholesterol. When there is little or no

cholesterol in the diet, synthesis is greatest; when the cholesterol in the diet is increased, however, synthesis is depressed and may become negligible. There seems, therefore, to be a mechanism controlling the total production of cholesterol.

Cholesterol may also be broken down and excreted. It has been shown that by labelling C4 and C26 the side chain of cholesterol can be broken down to carbon dioxide and water, but the ring part of the molecule is not broken down in this way. There is little evidence to indicate how the cholesterol nucleus is utilized in the animal body. Cholic acid is definitely formed, and perhaps cholesterol may also be converted to steroid hormones.

Cholesterol may be excreted in the bile, and most of it is reabsorbed. Some, however, in the form of dihydrocholesterol cannot be reabsorbed, and some is converted to coprosterol; so in these forms part of the cholesterol in the bile is excreted via the fæces.

Normally, therefore, there is an equilibrium between cholesterol synthesis and cholesterol destruction, and the amount of cholesterol synthesized by the liver is the fine adjustment. What controls this we do not know, but hypophysectomy in rats greatly reduces cholesterol synthesis. We also find that cholesterol synthesis is greatly increased in alloxan-diabetic rats and in chickens fed stilbœstrol, so that the controlling factor may be hormonal.

We therefore still know very little about cholesterol metabolism, but the data obtained from animal experiments suggest that we should endeavour to investigate further by radioactive tracer techniques the rate of synthesis of cholesterol and the excretion of cholesterol in patients with essential hypercholesteræmia.

DR. WHYTE: I cannot say why this patient's serum cholesterol was high, whether she was manufacturing excessive amounts of cholesterol or whether her destruction and excretion of it were impaired. It would have been valuable had we been able to carry out balance studies with radioactive tagging of cholesterol. Certainly her dietary intake was not excessive, for all her life she had eaten only small amounts of fat and cholesterol-containing foods. Thannhauser has made some attempt to classify the various causes of hypercholesteræmia, but it is impossible to do this satisfactorily in the present state of knowledge. He attributes primary hypercholesteræmia to excessive production by the liver. Schoenheimer, on the other hand, described a case in which the defect was on the other side, with impaired breakdown and excretion.

This is an example of the primary disease, because there is no evidence of any disease which might secondarily cause hypercholesteræmia. There is no history of jaundice or other indication of liver dysfunction, she is not myxœdematous, and there are no signs of any of those diseases which might cause hyperlipæmia in which the cholesterol level is raised as well as that of neutral fat—unless you think they were attacks of pancreatitis she suffered from. But it would be extremely unusual, I think, for the pain of pancreatitis to radiate down the arm. Whether pancreatitis occurring in association with hyperlipæmia is the cause or the result of the condition is uncertain. Perhaps it can be a complication of hypercholesteræmia; certainly there is encouragement for cholesterol gall-stones to form in this disease, and this might lead on to pancreatitis.

No, I think she had a coronary occlusion. However, severe pain in the back was such a prominent symptom that a dissecting aneurysm of the aorta has to be considered, especially as the femoral pulses were unequal, though this may have been due to local arterial disease. Perhaps she did have repeated small dissections of the aorta, and perhaps one episode resulted in obstruction to the mouth of the right coronary artery. At any rate the electrocardiographic tracings of September, 1954, are absolutely typical of a posterior myocardial infarction in spite of the differences of opinions given at that time. Lead 3 shows a Q3 and typical covering of an inverted T wave, and there are corroborative changes in leads 2 and aVF. The description of her attacks given here, including shock and radiation of pain, is quite typical of a coronary disaster with subsequent minor recurrences or repeated anginal attacks. The radiation of pain from one organ is sometimes influenced by the presence of an "irritable focus" in another organ. Coronary pain could perhaps be referred to the back because of coexisting disease of the aorta or pancreas or gall-bladder, organs which normally radiate pain to the back. But she had had infarction of the myocardium, and it was most likely a recurrence of this that killed her, though I should not discard the possibility of aortic dissection too readily.

Anyway, it is surely beyond dispute that she had atheroma, and I should be interested to know whether the radiologist can see any evidence of calcification in these radiographs.

Dr. P. Grattan-Smith: The radiograph of the chest is normal, and I can see no calcification. In the legs there are one or two flecks of calcification, which are probably along the line of the tibial vessels.

Dr. Whyte: Atheroma formation is a prominent feature of this disease of essential hypercholesteræmia. Adlersberg, in reviewing this topic, believes that the disease is transmitted as a dominant trait and in its full-blown homozygous form embraces hypercholesteræmia, atheroma deposition in the arteries and even in the valves and lining of the heart, corneal arcus, xanthelasma and tuberous and tendinous xanthomatosis. Those who have it in a milder, heterozygous form may show nothing more than a raised serum cholesterol level. Among 200 members of families which harboured this gene Adlersberg found that 60% had a high serum cholesterol level and 40% had coronary disease. It certainly seems to be dangerous to carry about with you excessive amounts of cholesterol. Once again I think that Dr. Courtice can contribute by telling us something of the association between atheroma and fat—not the physiological, but the pathological aspects of lipoid metabolism.

Dr. Courtice: Several theories have been postulated to account for the formation of atheroma in the intima of arteries. In recent years much attention has been focused on the lipids. Even though factors other than lipids may be responsible for the initiation of atheroma, there can be little doubt that lipids *per se* are involved in the later stages.

The atheromatous lesions consist largely of lipid, which is present in much the same proportions as in the plasma. There is a good deal of evidence which supports the view that the lipids are implicated in atherosclerosis. I shall give some of this evidence, with which most of you are no doubt familiar.

In man the incidence of atheroma is lower in those on a diet of low fat and low cholesterol content. For example, in Oriental countries the incidence of atheroma is low in the cereal-eating poor people, and during the last war the incidence of heart disease decreased in Norway and Finland when the fat and calorie content of the diet decreased, but not in America and Sweden, where the diet remained the same as pre-war.

In certain experimental animals, such as rabbits and chicks, increased cholesterol in the diet produces hypercholesteræmia and atherosclerosis. In the dog this also occurs if thyroid activity is at the same time depressed. Giving œstrogens to birds causes hypercholesteræmia, and atheroma develops.

In certain diseases in man, such as diabetes, nephrosis and essential familial hypercholesteræmia, in all of which there is a disturbance of lipid metabolism, with high serum cholesterol levels, the formation of atherosclerosis is accentuated. It seems, therefore, that in these instances at any rate, lipids and especially cholesterol are implicated in the genesis of atheroma.

The next question to ask is whether cholesterol in any particular form is responsible. Is it just cholesterol *per se*, or is it some special combination of cholesterol? Gofman has fractionated plasma lipids by means of the ultracentrifuge. By increasing the specific gravity of the plasma, the lipids will be made to float to the surface on centrifugation. The ease with which these particles float depends partly on their size and density. In this way Gofman separates lipids according to their Svedberg flotation units into groups such as Sf greater than 70, Sf 30 to 70 and Sf 3 to 8. He claims that those individuals with a high Sf 10 to 20 value are prone to atherosclerosis.

It may be that the amount of a certain lipid combination such as exists in the Sf 10 to 20 group is responsible. This may be bound up with the cholesterol-phospholipid ratio. If the cholesterol-phospholipid ratio is high, the cholesterol may be more likely to go out of solution and be deposited.

This may be significant in man. Man has a preponderance of β lipoprotein, which has a higher cholesterol-phospholipid ratio than α lipoprotein. In animals most of the cholesterol is present as α lipoprotein, but in rabbits fed cholesterol it is the β lipoprotein that increases.

Overall evidence, therefore, suggests that lipid metabolism or some disorder of it is implicated in the genesis of atherosclerosis. In this case which Dr. Whyte is discussing there is hypercholesteræmia and, I should think, a high Sf 10 to 20 value and a high cholesterol-phospholipid ratio; so one would expect to find extensive atheroma of the arteries.

Dr. Whyte: There is no doubt in my mind that in this and similar cases the disturbed state of fat metabolism has led to the production of atheroma. The patient behaved very much like a cholesterol-fed rabbit. Now just a word or two about her treatment. She had thyroid, heparin, œstrogen and a low-fat diet at various times. Thyroid influences cholesterol metabolism, as we have heard, but it does but little good in hypercholesteræmic conditions unless hypothyroidism is the cause. It was of no noticeable value in this case. Heparin has had a remarkable career in recent years, quite apart from its being an anticoagulant. It sometimes gives immediate relief of coronary pain, apparently due to vasodilatation, but the results of controlled trials lend no support for the practice of giving occasional injections of heparin, perhaps weekly or fortnightly, as prophylactic therapy in angina. But another interesting effect of heparin is its ability, when injected *in vivo*, to abolish or at least to diminish the milkiness of serum resulting from a fatty meal. Abnormal fat patterns are said to change towards normal, and heparin injections retard the formation of atheroma in rabbits fed on cholesterol.

The rationale of œstrogen therapy is based on the known relation between atheroma and sex. Coronary disease, and atheroma in general, are less common among females than males till after the menopause. Œstrogen treatment of males, as in patients with carcinoma of the prostate, is said to reduce the degree of atheroma to the level of females, and the reverse is said to occur when females with carcinoma of the breast are treated with testosterone. The exact mode of action of œstrogens or their efficacy as a mode of treatment in these conditions is not known. It is difficult to say whether œstrogen treatment helped her, though there is a suggestion that the xanthomatous deposits decreased. This may have been partly due to the dietary restriction. To reduce the intake of cholesterol and fats helps to relieve some of the strain on cholesterol-handling, it is advised by all authorities in this condition, and it sometimes reduces the serum cholesterol level, though not to normal. This means that the patient must become a vegetarian.

Let me now summarize this story. Clinically, this patient suffered, I think, from essential hypercholesteræmia. She presented with xanthomatosis, she developed arterial disease in her legs causing intermittent claudication, and she died at the age of forty-five from coronary occlusion. Physiologically, she suffered from the effects of a disturbance in lipoid metabolism which led to excessive amounts of cholesterol being present in the serum and in the skin and blood vessels. The fundamental cause of the disturbance is not known: perhaps it was excessive synthesis of cholesterol, or, more probably, defective destruction or excretion. Pathologically, I think we can expect Dr. Palmer to demonstrate cholesterol-laden lesions in the skin, in the blood vessels, perhaps even in the endocardium, and in other viscera; an old posterior myocardial infarct and perhaps a recent one with an obstruction in a coronary vessel; perhaps some dissection of the aorta; perhaps a gall-stone. And it would complete the story very nicely if he could show us the genes which have caused all this fuss.

Dr. Ross: Dr. Mervyn Matheson was invited along today. He did a lot of hard work in looking after the patient in between her visits to the hospital. Dr. Matheson, do you think this was a clear presentation of the patient's history and do you agree that that is the most likely diagnosis?

Dr. M. W. Matheson: Yes, I do. My association with her was in her home, attending her for the severe coronary pains. They were really severe but responded to pethidine. In 1954 she had a series of them. I had not seen her from that time until the night of her death. She was dead when I arrived. She had gone out to the sink and collapsed; her husband heard her fall and said she breathed stertorously for twenty or twenty-five minutes and then died. Leading up to that she had apparently had several very severe attacks of coronary pain the day before.

Dr. Ross: We want to thank Dr. Matheson, an old student of Sydney Hospital, for taking such an interest in this patient and arranging for the post-mortem examination to be done here. Now Dr. Finley, I believe, saw this patient in the hospital. Would you care to say something?

Dr. A. G. Finley: Actually I saw this patient for some time prior to the onset of the cutaneous manifestations of this malady. I had treated her on two or three occasions for a skin eruption, which I regarded as having no connexion with her essential xanthomatosis. She came to the skin out-patient department with pustular lesions on her hands and responded satisfactorily to treatment each time. It is interesting that the last episode of this pustular condition cleared only a matter of two months before she returned to

show me her palms and asked: "What is this?" Certainly the cutaneous manifestations of this metabolic disorder had progressed very rapidly, as she had had no evidence of the yellow discoloration two months previously.

From a dermatological point of view it is interesting to speculate as to why these people get deposition of xanthomatosis material on certain areas of the skin. There are various patterns of xanthomatosis which we recognize, which have been indicated by Dr. Whyte, and each of these patterns has a fairly characteristic distribution. It is tempting to believe that there may be some local tissue factor in the skin which is responsible for the deposition of cholesterol. What it is, I do not know. It is suggested that it might have some relation to trauma or pressure, but I do not think there is any substantial evidence at the moment to indicate exactly what it is.

Dermatological thinking about xanthomatosis has, I think, been impeded till recent years, because dermatologists of the last century were largely concerned with the recognition of various morphological patterns on the skin. This applied to xanthomatosis. They classified xanthomata according to their physical appearances, and, allowing for the fact that they did not have the biochemical knowledge available to us today, they had no conception of the ætiology of these conditions. Today text-books produce classifications which are based to some extent on the morphology of these eruptions and to some extent on ætiological considerations. I believe that the modern classifications given by people like Thannhauser, based on biological, genetic and clinical observations, are much more logical. At the same time that is not to say that the recognition of the pattern on the skin is not without value. It is recognized, for example, that *xanthoma tuberosum* is almost invariably associated with a raised blood cholesterol level, whereas *xanthoma disseminatum* is not, and there are other correlations of that sort.

DR. ROSE: I see Dr. Wilfred Evans here today. I think, Dr. Evans, this is a subject you used to be interested in.

DR. WILFRED EVANS: My chief interest in this case is a clinical one, and, as Dr. Whyte has stated, the differential diagnosis of the cause of death lies, I think, between pancreatitis, dissecting aneurysm and coronary thrombosis with myocardial infarction. I think pancreatitis is unlikely because the patient did not have the acute lower back pain which is so characteristic of the condition. The question of dissecting aortic aneurysm is difficult to exclude, as Dr. Whyte has said, but I think it is unlikely, because the pain is typical of the coronary type and there was no evidence of an aortic regurgitant murmur, which is usually present in aortic dissecting aneurysm. So I think it is most probable that she died from a coronary occlusion, for, as Dr. Whyte pointed out, she had already had one myocardial infarction, and also she had evidence of generalized arterial degeneration as shown by the intermittent claudication and the previous infarction. In regard to the other question, the general question of causation of atherosclerosis, which Dr. Courtice has so ably dealt with, this subject was discussed last year at the cardiological meeting in Washington. A very great deal of work is being done on it, and people all over the world are assessing how much atherosclerosis is affecting different populations. It was quite striking to hear that the amount of atherosclerosis occurring in Minnesota—Americans are great eaters of fat, of course—was assessed at forty times that which is found in a fishing village of Kyushu in Japan. So the intake of total fat (and the emphasis was laid more on total fat than on cholesterol) appears to have a distinct bearing on the incidence of atherosclerosis. There was one other aspect which Dr. Whyte dealt with, and that was the question of œstrogen. A lot of people in the States were keen on giving œstrogen because of the fact that women below the age of the menopause seldom develop atherosclerosis, but after trying it on a number of men, the men decided they would prefer to have the atherosclerosis!

Autopsy Report.

DR. A. A. PALMER presented the following autopsy report:

The body was that of a well-nourished, well-developed female. The lesions on the elbows and palms of the hands were much less evident at post-mortem examination than they were during life.

Thyroid. This was rather small. The thymus was not found.

Lungs. These were normal.

Heart. The left ventricle was moderately dilated; it was not hypertrophied. There was gross atheroma of both coronary arteries and their branches with severe narrowing

in many places, most marked near the origin of the left coronary artery, where a cross section showed much soft yellow friable material in the wall. No recent thrombus was found. The valves were normal. The thoracic aorta showed only slight atheroma. There was severe atheroma of the distal three inches of the abdominal aorta and common iliac arteries. The renal arteries were not affected.

Liver. This was diffusely congested.

Spleen. This was congested.

Kidneys. These were congested.

Œsophagus, stomach, intestines, pancreas, suprarenals. No abnormality was detected in these.

Pelvis, bladder, genital organs. The uterus had been removed. The right ovary was atrophic.

Brain and spinal cord. The cerebral vessels were practically free from atheroma; there were a few patches in the basilar and middle cerebral arteries. The cerebrum, brain stem and cerebellum were normal. In the pituitary no abnormality was detected.

Microscopic Examination.

Arteries. Section of aorta, coronary arteries and posterior tibial artery confirm the presence of advanced atheroma.

Heart. There is extensive myocardial fibrosis.

Thyroid, adrenal, pituitary, pancreas, spleen, liver and kidneys. These show no significant changes.

Skin. In the affected region there are a few lipoid-laden cells and the adjacent dermis is abnormally cellular, suggesting a xanthoma from which much of the lipoid has disappeared.

Diagnosis.

Essential hypercholesterolæmia with advanced coronary artery atheroma and myocardial fibrosis.

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Out of the Past.

In this column will be published from time to time extracts, taken from medical journals, newspapers, official and historical records, diaries and so on, dealing with events connected with the early medical history of Australia.

MEDICAL OFFICERS OF TRANSPORTS.[1]

[From "An Historical Journal of the Transactions at Port Jackson and Norfolk Island", by John Hunter, Esq., Post Captain in His Majesty's Navy, London, 1793.]

Cape of Good Hope, January, 1789.

I WAS now very anxious to get some account of the transports, which under the command of Lieutenant Shortland, the agent, had left Port Jackson on the 14th of July, 1788, and which I was sorry to understand had not been in this bay: for I thought it mighty probable, that as their route was to the northward, by the Molucca Islands and Batavia, they would certainly touch here on their way home. It being now seven months since they sailed I was apprehensive for their safety, particularly when I considered the very weakly condition of some of the crews, by the scurvy, when they left us, and not a surgeon in any one of the ships. This must be allowed to be very improper economy in the owners of these ships, when the extent of the voyage they had undertaken is considered, together with the well known impossibility of their being able to procure seamen, as any recruit of strength to their ships companies in that inhospitable and far distant part of the world—I cannot help here taking the liberty of saying, that it is much to be lamented, that when ships are hired for the service of the Government, to perform such long and trying voyages to the health of those employed in them, that it is not made a part of the contract and practice that they carry a surgeon: for I know well, that seamen, when taken ill upon such long passages, are, at the very idea of being without the assistance of a surgeon (although careless and void of thought at other times when in perfect health) apt to give way to melancholy, and a total dejection of spirits: and that many a valuable subject has been lost to the country by such a trifling

[1] From the original in the Mitchell Library, Sydney.

saving. Out of the nine transports which were employed on this service, one only had a surgeon: and that one, had she not been bound upon some other service, after leaving Port Jackson, would in all probability have been without one also.

Obituary.

JOHN BERCHMANS DEVINE.

THE death on October 31, 1955, of Dr. John Berchmans Devine at the early age of forty-two years deprived Australian surgery of a thoughtful and enterprising man who had already made important contributions to surgical practice. As a small boy he loved to see "how the wheels go round", and the visitor to the Devine household, if he was interested in the doings of small boys, had to watch his step in the small upstairs room which was crammed full of wires and gadgets set up with the hope or the intent that something would happen. As the boy grew to manhood and took up the study of medicine his mind continued to inquire and his fingers to experiment.

The son of a famous father, John Devine started his education at Xavier College, Melbourne, continued it at Beaumont College, England, and then became a student in the faculty of medicine at the University of Melbourne. He did well in his course—he graduated M.B., B.S. in 1937, taking first class honours in medicine and gaining the Jamieson Prize in that subject; he also obtained first class honours in surgery. He was placed second in the honours list of his year; he was appointed a resident medical officer at the Royal Melbourne Hospital and later on one of its registrars. In 1940 he obtained the degree of Master of Surgery at the University of Melbourne. Soon after that he enlisted in the second Australian Imperial Force and sailed for the Middle East. He was in Tobruk throughout its siege and for his service there was mentioned in dispatches. It was here, of course, that he gathered the data for his book "Rats of Tobruk". Some of the illustrations in this book were taken from drawings and oil sketches made by him on the spot. In the foreword General Blamey wrote: "Tobruk will be linked for ever with the 2nd A.I.F. by the intimate story told by Major John Devine."

After being demobilized, John Devine returned to civil practice. He obtained the Fellowship of the Royal College of Surgeons of England and also the Fellowship of the Royal Australasian College of Surgeons. He was appointed to the honorary out-patient staff of the Alfred Hospital, Melbourne, and later to that of the Royal Melbourne Hospital. That his versatile and inquiring mind helped him in his surgical career is clear from the many contributions that he made to surgical literature. He was enthusiastic and always active. He kept abundant clinical notes, and the photographic records of his surgical work were first rate. He was good to his patients and never thought of sparing himself in their service. One tale that is typical of him may be told. An old man, a farmer, was faced with a serious operation, but would not have it done because he had left his cows in their yard and had made no arrangements to have them fed and milked. The farm was twenty-five miles out of Melbourne. Devine got into his motor-car and went out to the farm, where he arranged for the cows' welfare. The old man's operation was successfully performed on the same day.

John Devine excelled in surgery of the gastro-intestinal tract. He was expert in the use of the gastroscope and of the Miller-Abbott tube. With his father, he was co-author of "The Surgery of the Rectum and Colon". His enthusiasms carried him beyond surgical practice and the devising and making of surgical instruments for his own use. Photography and cabinet-making claimed his attention and he excelled at both. One would think that here was a young and successful surgeon, gifted as few young men are, and with a still brighter future. But well over three years ago his health broke down, seemingly from an intensity of overwork on top of the remote effects of war. The dominant features of his illness were a transient phase of functional nervous symptoms, a loss of energy and of resistance to infection, an intractable chronic infection of antral origin with bony involvement, persisting with pain and misery, which many operations could neither cure nor ameliorate. He died of a coronary occlusion. His death was unexpected, as for some time before he died he had sufficiently recovered to do part-time research, and at the time of his death he had been happily working for four months. He is the twenty-third member of those "Rats of Tobruk" who went

through the famous siege to die this year in Victoria between the ages of forty and fifty and whose deaths can be largely attributed to their Tobruk experiences.

In the heyday of his health and happiness John Devine was a bright, well-informed and cultured man who radiated energy and good will. Those who held him in high regard will grieve at his early death, and much sympathy will be extended to the members of his family.

DR. W. D. G. UPJOHN writes: Throughout the ages thoughtful men have reflected on the brevity of human life and have stressed the fact that what is most valuable is not the length of a man's life but its quality.

John Devine died at an early age when most surgeons are considered to be in the most significant and active period of their professional lives, but he had already lived more fully than many a man who has reached the allotted span of life.

His most outstanding characteristic was his intense intellectual activity. When a difficult task was presented to him he displayed an astonishing energy in his endeavour to solve
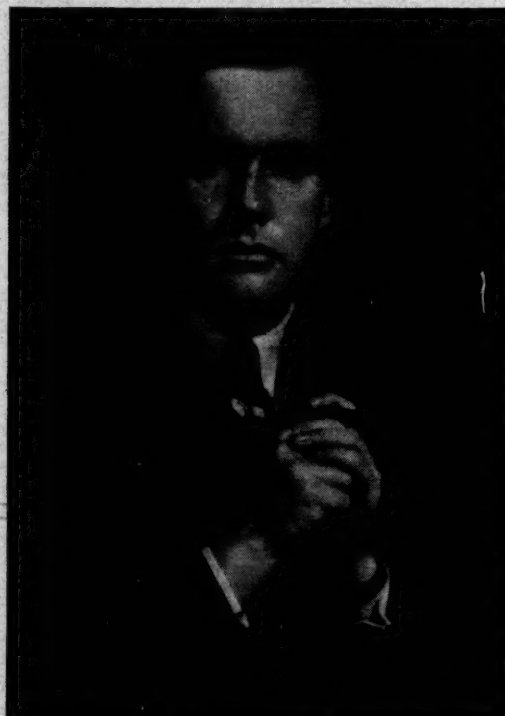
it. When a new idea occurred to him, he worked on it with a concentration which enabled him to ignore unrelated or less important interests.

When his mind was thus occupied, his thoughts might come too quickly for adequate expression in words. This made it difficult at times for his hearers to understand the course of his thoughts, but when he came to write down the matured product of his thoughts, observations and conclusions, he expressed himself admirably. Examples of this can be found in his various publications.

In addition to his great enthusiasm in surgery he was very much interested in art and literature, and he showed a considerable talent for sketching and painting. Many must have read his book on his war experience in Tobruk. It is most entertaining reading.

He became interested in colour photography and the making of colour films on various subjects not confined to surgery. They showed a painstaking attention to technique. In fact he seemed to take a delight in encountering and overcoming technical difficulties whether they were in surgery, experimental physiology or photography. The greater the difficulty, the greater the zest he displayed in mastering it.

He showed remarkable ingenuity in devising instruments and technical devices, and these he generously communicated to his fellow workers.

As a student, resident medical officer and mature surgeon he was never lukewarm in what he did. He put all he had into the task he had in hand, not in a grim way, but with a youthful zest and imaginative expectancy.

Even during recent years when it was obvious that he was ill and should have rested himself, he worked with an intensity that would have fatigued a man in good health. One could almost imagine that he had some intuition that his activities would cease prematurely, and that he was driven by this inward sense of urgency to perform his life's task before it was too late.

Amidst all this tremendous activity he was never too busy to spare time to teach his students and nurses. To the latter he gave the most detailed and lucid instructions in writing about what had been done to the patient, and what was the nurse's part in helping bring about a successful result. Many nurses and younger medical graduates speak in grateful memory of his kindly help and of his patient explanations of details in the diagnosis and treatment of his patients. He was always ready to come to the aid of his young colleagues.

He will be remembered as a bright, active-minded and kindly young surgeon. He accomplished much, and he had great potentialities which did not reach full fruition because of his illness and too early death.

EMERITUS PROFESSOR W. A. OSBORNE writes: John Devine as a student was of that type which brings joy to the heart of the teacher—alert, keen, responsive and thorough, but always modest. Later when he was carrying out some research of his own designing in my laboratory, I was able to see and admire his courage and resource in overcoming experimental difficulties and generally in his mastery of complicated techniques. I hoped he would devote his life to the scientific side of medicine and especially in a university where his infectious enthusiasm, originality of mind, attractive personality and wide culture would inspire students, but the more directly human aspect of surgical practice had a deeper appeal and determined his career. Charm is difficult to define and more difficult to analyse, but it never fails to make its presence felt by others; different observers single out different components to which they react; one such with me was his modulated and very pleasing speaking voice. Alas, that all these winning qualities are now a memory only!

Correspondence.

A SURVEY OF RUBELLA PREGNANCIES.

SIR: The annotation entitled "A Survey of Rubella Pregnancies" which appeared in THE MEDICAL JOURNAL OF AUSTRALIA on February 4, 1956, on page 185, tends to create an erroneous impression with regard to my views on the incidence of congenital malformations following maternal rubella in pregnancy.

Actually (in 1943, not in 1946, as stated in your journal) I wrote (Swan et alii, 1943):

> On the available evidence, when a woman contracts rubella within the first two months of pregnancy, it would appear that the chances of her giving birth to a congenitally defective child are in the region of 100%, and if she contracts rubella in the third month they are about 50%.

That this conclusion was merely tentative is indicated by the terms in which it was couched.

With greater experience during the course of the five years of the South Australian investigation and with the advent of information from other workers, it became evident that the original view would have to be modified. This modification (again based on the evidence then at my disposal) is embodied in the essay (Swan, 1949) awarded the Katherine Bishop Harman Prize of the British Medical Association for 1949.

Incidentally, mention is made in this essay of various difficulties encountered in attempting to assess accurately the risk to the foetus in a pregnancy in which the mother develops German measles.

Nevertheless, the problem is not an insoluble one. It is hoped that there will be a widespread and enthusiastic response to the investigation which is being carried out under the ægis of the Department of Obstetrics and Gynæcology of the University of Melbourne, so that we may eventually arrive at the truth.

Yours, etc.,

CHARLES SWAN.

175 North Terrace,
Adelaide,
February 8, 1956.

References.

SWAN, C., TOSTEVIN, A. L., MOORE, B., MAYO, H., and BLACK, G. H. BARHAM (1943), "Congenital Defects in Infants following Infectious Diseases during Pregnancy: With Special Reference to the Relationship between German Measles and Cataract, Deaf-Mutism, Heart Disease and Microcephaly, and to the Period of Pregnancy in which the Occurrence of Rubella is followed by Congenital Abnormalities", M. J. AUSTRALIA, 2 : 201.

SWAN, C. (1949), "Rubella in Pregnancy as an Ætiological Factor in Congenital Malformation, Stillbirth, Miscarriage and Abortion", J. Obst. & Gynæc. Brit. Emp., 56 : 341, 591.

THE TREATMENT OF ULCERATIVE COLITIS.

SIR: In his article (M. J. AUSTRALIA, January 28, 1956) on the treatment of ulcerative colitis, Mr. E. S. R. Hughes gives us a very convincing exposition of the benefits of surgery in advanced cases of this perilous condition. Through the perseverance of surgeons and their achievement of a tolerable ileostomy, many cases can now be restored to reasonable health. The key to this success, and it is the landmark in treatment, is the modern ileostomy.

With chagrin, I recall a patient in whom an ileostomy allowed the ulcerative colitis to resolve completely in 1945. However, the dysfunction of the ileostomy was so unbearable that she insisted on the restoration of her alimentation, "come what may", maintaining that she would prefer the worst stages of diarrhœa and even risk death.

The mental attitude of this patient brings me to the point of this letter, which is to contradict Mr. Hughes in his statement about the role of disordered psyche in the ætiology, and of psychotherapy in the treatment of ulcerative colitis. When he talks authoritatively of surgical problems, I am very favourably impressed. When he states, "A personal and regular review of 18 patients with ileostomy has failed to reveal any abnormality in personality, an experience similar to that of Counsell and Lockhart-Mummery (1954)", it is a different matter. He reminds me of an eminent surgeon who examined a traumatic neurosis with me recently. It was an accident case, and we were representing opposing interests. The patient was female, elderly, garrulous, ignorant and irritating. I had written hardly four lines of history when he shouted at her that she was not addressing the jury yet. After dismissing her as just neurotic, which was, of course, correct without the "just", and haranguing me about the evils of neurosis, and what terrible people they were, he started to tell me about his own peptic ulcer. Excellent though his surgical skill, he is not qualified mentally, emotionally or academically to recognize, understand or treat neurosis. From experience, I am sorry to say, the same applies to most surgeons and to the majority of the profession. This is a sweeping statement of the type that begets controversy and antagonism, but unfortunately it is true.

The opprobrium associated with neurosis is a telling measure of our attitude to the disease. "Neurosis" has become a term of contempt with both the profession and the general public. Since I started this letter, a colleague has been in touch with me about a patient with a frozen shoulder syndrome whom he had discharged from hospital prematurely after a long, exasperating period of trial for everyone, especially the hospital staff. "He is just neurotic", was his final remark.

We are hostile to neurosis because we are frustrated and defeated by it. It has confounded us in diagnosis and defied us in therapy. The hostility is, of course, subconscious. We will be the first to admit that we do not understand the disease. Special and long training is essential to gain the limited knowledge we have of neurosis. These facilities are not available to most doctors. The long and detailed surgical training seems to preclude it for most surgeons.

I write to contradict Mr. Hughes on this point of the ætiology because he will be quoted in the future, just as he has quoted other eminent authorities (Counsell and Lockhart-Mummery, 1954).

It is a pity he did not quote Counsell and Lockhart-Mummery *verbatim*. It would help to keep things in their right perspective. "We suspect that too much emphasis may have been placed on the emotional factor in the ætiology of this disease. We believe that a psychiatric study of patients successfully treated surgically would be instructive." I note in their article that over 20% of the married women (fourteen) had not yet resumed normal relations (sexual) "for reasons which appeared to be psychological". Nevertheless, the impression given by Mr. Hughes is that there was no abnormality of personality.

I have treated cases of ulcerative colitis for nearly twenty years, and I have had a special interest in neuroses during the greater part of this period. For the past twelve years, neuroses have constituted at least 70% of my hospital and private cases. After a period, the pattern of neuroses becomes as clear-cut as shock to the surgeon, a Tschaikovsky composition to a musician, or a Renoir to a painter. This distinctive pattern of neurosis has been present in every case of ulcerative colitis that I have seen. In my opinion, it has been fundamental as regards ætiology. Factors as diverse as the castor oil induction of labour, emotional shock, emotional frustration and antibiotics may precipitate the attack, but it is the basic neurosis which prevents the initial inflammatory reaction passing through the normal local process of healing, and leads to the organic complications of the psychosomatic state we know as ulcerative colitis.

To argue that emotional factors are not causative as psychotherapy does not cure, is similar to arguing that a bullet was not causative as its removal did not cure the optic nerve blindness.

Over the years I have treated a number of cases with indifferent results. There have been a number of "cures", a number of failures, but definitely no mortality. One of my failures was "cured" subsequently by medical treatment at the hands of another physician. As regards the danger from psychoanalysing these patients, psychoanalysis in the strict sense of the word is not necessary in the great majority of cases. I find I can get an adequate appraisement of their psyche in two interviews in most cases. An interview with a close relative and observation over a week has been ample. As regards psychotherapy, which Mr. Hughes decries, I see he states: "They should be reassured that there is no malignant disease; they should be told that they can be cured, but that this may mean operation." This is definite psychotherapy, even if of a horse and buggy standard. It will be a pity if the surgical treatment of ulcerative colitis is jeopardized by a wave of surgical enthusiasm. A similar tragedy has occurred in another psychosomatic state. It is hoped that the sufferings, frustrations and deaths of countless recipients of gastro-enterostomies, varied gastrectomies and vagotomies will not be lost in the archives of therapeutic endeavour.

In conclusion, I submit that every case of ulcerative colitis should be seen by a psychiatrist or equivalent specialist within a few weeks of diagnosis. If no substantial progress occurs within a month, they should be referred to a physician with a special interest in ulcerative colitis. Ileostomy should be considered if they have not substantially recovered after two months, or if the patient deteriorates rapidly at any time.

Yours, etc.,

BRIAN HAYNES.

185 Macquarie Street,
　Sydney,
　　February 14, 1956.

THE DEVELOPMENT OF MODERN PSYCHIATRY IN RELATION TO MEDICINE AND SURGERY.

SIR: To understand the mind and how it works is a problem of the greatest interest and importance to mankind. The progress made in recent years in many branches of science and medicine has been far beyond what one would have thought possible beforehand. This encourages the belief that if sufficient interest can be directed to the study of intellectual processes, they may, as has been the case with other apparently unsolvable problems, yield up their secrets to those interested. By influencing others to think about this subject, anything drawing attention to it may help to bring about the ultimate object.

I was therefore pleased to see in Dr. McLaren's letter (M. J. AUSTRALIA, February 4, 1956) that the subject matter of my Stawell Essay has raised for him the question of the relationship between the functioning nervous system and the functioning mind. In addition, Dr. Jansen's letter (M. J. AUSTRALIA, December 3, 1955) carries reference both to the central nervous system and to mental activity. At least the essays help in this small way, but the important question of how the mind interprets impulses is still as far from being answered.

The impressed neuron theory may be a slight advance towards this object, but it can only be regarded as an extension of what is already known about exteroceptive impulses.

Exteroceptive impulses, arising as the result of extra-cerebral incidents, form an intracerebral record of the incident, by means of neuronic impression. Impressed neurons are given value to the mind by the associations made with them.

The ultimate effect of impulses on the mind, whether acting directly as freshly produced exteroceptive impulses or indirectly through the established connexions of impressed neurons formed from old exteroceptive impulses, appears to be due to the association which has been previously made with that class of impulse. Meaning to the mind or value placed on impulses bearing some characteristic impression is given in this way.

Taking Shelley's "Ode to a Skylark" to illustrate this point, to Dr. McLaren it is a thrilling joy, but to the person who as a nervous child was soundly thrashed several times because he was unable to quote it, it is a trembling horror. Further, to a true native of Australia never taught to read or write it would be but black on white, not without any meaning, but perhaps without any meaning we would understand. This suggests that after neurons are impressed, then in accordance with the individual's mental make-up, they are associated with certain feelings or emotions previously related to a similar environmental incident, but it does not show us how these are interpreted by the mind.

At birth there are a minimal number of neurons impressed with exteroceptive impulses. Because these are insufficient to enable the mind to function, there can be no intellect and no meaning. At this stage, the brain may be compared to a gramophone without any record disks. During the months following birth, neuronic records are being formed, classified and associated with feeling and emotion or something to which the brain is able to attach some meaning. When sufficient neuronic records and associations have been made, meaning follows. Intellectual expansion seems largely to be an additive process, which continues throughout life in a manner similar to that by which the earliest impressions were dealt with.

There will be many different ideas put forward to explain the way in which the mind functions, but until this problem is fully understood, it will not be possible to know which ideas are right, nor to be definite about the relationship of the brain and mind.

My essay is concerned only with the materialistic side of this matter. It does not, by itself, extend far enough to even suggest how the mind functions. But the impressed neuron postulate, so far as it goes, is in keeping with Dr. McLaren's theory, for it shows how man's responses and behaviour are influenced by environmental circumstances, and that through the environmental recording section of his brain he is attuned to the cosmos. (How this has come to pass and why is briefly indicated in the Stawell Essay, and I hope to make these points less obscure in a later article.)

I am not sufficiently versed in the part played by spiritual factors in mental processes to make further rational comment on Dr. McLaren's synthesis. I would, however, commend him on stating his understanding of the brain-mind relationship, because, whether right or wrong, it offers something definite for consideration. If found acceptable, it should be examined for extension and application; if not, then by a process of addition, subtraction, substitution and alteration, a more satisfactory explanation may be reached through it.

Further comment from anyone interested in this important matter, brought forward by Dr. McLaren, might be of service in its elucidation.

Yours, etc.,

H. B. RUDDUCK.

940 Nepean Highway,
　Moorabbin,
　　Victoria.
　　　February 13, 1956.

Australian Medical Board Proceedings.

NEW SOUTH WALES.

THE following additions and amendments have been made to the Register of Medical Practitioners for New South Wales in accordance with the *Medical Practitioners Act, 1938-1955*:

Registered medical practitioners who have complied with the requirements of Section 17 (3) and are registered under Section 17 (1) (*a*) of the Act: Potts, John Louis, M.B., B.S., 1950 (Univ. Adelaide); Reaburn, Robert John, M.B., B.S., 1953 (Univ. Melbourne); Cooper, Robert Gavine, M.B., B.S., 1954 (Univ. Queensland).

Registered medical practitioners who have complied with the requirements of Section 17 (3) and are registered under Section 17 (1) (*b*) of the Act: Boxall, Betty Bryan, M.R.C.S. (England), 1950, L.R.C.P. (London), 1950, M.B., B.S., 1950 (Univ. London); Fisk, Graham Chudleigh, B.M., B.Ch., 1953 (Univ. Oxford).

Registered medical practitioner who is required to complete twelve months' hospital service in accordance with the provisions of Section 17 (3) and is registered under Section 17 (1) (*a*) of the Act: Godfrey, Robert Lindsay, M.B., B.S., 1955 (Univ. Melbourne).

Registered medical practitioner who is required to complete twelve months' hospital service in accordance with the provisions of Section 17 (3) and is registered under Section 17 (1) (*c*) of the Act: Zawadzki, Irene.

The following additional qualifications have been registered: Schiller, Eric, M.R.C.P. (London), 1955, M.R.C.P. (Edinburgh), 1955; Coppleson, John Victor Malcolm, M.R.C.O.G. (England), 1954; Thwaite, Thomas John, F.R.C.S. (Edinburgh), 1955, F.R.C.S. (England), 1955; Morrissey, Matthew John, D.O., 1955 (Univ. Sydney); Lamond, Paul Kenneth, D.C.P., 1955 (Univ. Sydney); Stuckey, Douglas Seavington, M.D., 1956 (Univ. Sydney).

Post-Graduate Work.

THE POST-GRADUATE COMMITTEE IN MEDICINE IN THE UNIVERSITY OF SYDNEY.

General Revision Course.

THE Post-Graduate Committee in Medicine in the University of Sydney announces that the annual general revision course will be held for two weeks from Monday, May 7, to Friday, May 18, inclusive. The main theme of the course is therapeutics, and programmes will be available shortly.

As in former years, the programme will be a comprehensive survey of modern trends in diagnosis and treatment of special value to general practitioners, consisting of lectures, special demonstrations, symposia, discussions and conferences. Four half-day sessions have been devoted to general medicine, two half-day sessions each to pædiatrics, psychological medicine and surgery, and one each to anæmias, dermatology, gynæcology, obstetrics and neurology.

Special demonstrations will be held on blood typing and transfusion and on clinical laboratory methods. An X-ray conference will also take place, to which members of the course are invited to bring their own X-ray films for discussion. In addition, lectures and demonstrations will be given on modern methods of anæsthesia, diabetes, new drugs and therapeutic measures.

Social activities will include a cocktail party and a theatre party, and the ninth Annual Post-Graduate Oration will be held in the Great Hall of the University of Sydney, to which all members of the course and their wives will be invited. A golf competition for the Post-Graduate Golf Cup, open to members, will also be held, and the Brydon Cup will be for competition by country members.

The fee will be £12 12s. for full-time attendance or £6 6s. for one week or part-time attendance. New Zealand and interstate applicants requiring accommodation in Sydney during the course will be given every assistance. Early written application, enclosing remittance, is essential and should be sent to the Course Secretary, The Post-Graduate

DISEASES NOTIFIED IN EACH STATE AND TERRITORY OF AUSTRALIA FOR THE WEEK ENDED FEBRUARY 4, 1956.[1]

Disease.	New South Wales.	Victoria.	Queensland.	South Australia.	Western Australia.	Tasmania.	Northern Territory.	Australian Capital Territory.	Australia.
Acute Rheumatism	3(1)	1(1)	4(3)	..	1	..	..	..	9
Amoebiasis	..	..	..	..	..	..	..	..	..
Ancylostomiasis	..	..	..	..	..	..	1	..	1
Anthrax	..	..	..	..	..	..	..	..	..
Bilharziasis	..	..	..	..	..	..	..	..	..
Brucellosis	1(1)	..	..	..	..	..	..	..	1
Cholera	..	..	..	..	..	..	..	..	..
Chorea (St. Vitus)	..	1(1)	..	..	..	..	..	..	1
Dengue	..	..	..	..	..	..	..	..	..
Diarrhœa (Infantile)	10(6)	14(10)	4(4)	..	2(2)	..	..	1	31
Diphtheria	..	3(3)	..	..	1(1)	..	..	..	4
Dysentery (Bacillary)	..	..	13(12)	1	1(1)	..	..	..	14
Encephalitis	..	1(1)	..	..	..	..	..	..	1
Filariasis	..	..	..	..	..	..	..	..	..
Homologous Serum Jaundice	..	..	..	..	..	..	..	..	..
Hydatid	..	..	..	..	..	..	..	..	..
Infective Hepatitis	195(122)	99(44)	..	19(5)	6(2)	4	1	2	326
Lead Poisoning	..	..	..	..	..	..	..	..	..
Leprosy	..	..	..	..	..	..	..	..	..
Leptospirosis	..	..	1	..	..	..	..	..	1
Malaria	..	..	..	..	..	..	..	..	..
Meningococcal Infection	7(2)	5(5)	..	..	..	..	..	..	12
Ophthalmia	..	..	..	..	..	..	..	..	..
Ornithosis	..	..	..	..	..	..	..	..	..
Paratyphoid	..	..	..	..	..	..	..	..	..
Plague	..	..	..	..	..	..	..	..	..
Poliomyelitis	8(4)	2(1)	5(3)	4(2)	41(28)	..	..	..	60
Puerperal Fever	..	..	..	1	..	..	..	..	1
Rubella	..	43(34)	1	3(1)	1(1)	..	..	1	49
Salmonella Infection	..	..	..	..	..	..	..	..	..
Scarlet Fever	4(2)	3(2)	2(1)	..	..	..	..	..	9
Smallpox	..	..	..	..	..	..	..	..	..
Tetanus	..	..	..	..	1	..	..	..	1
Trachoma	..	..	..	..	4(1)	..	..	..	4
Trichinosis	..	..	..	..	..	..	..	..	..
Tuberculosis	23(15)	17(16)	13(9)	2(2)	2(1)	2(1)	2	..	61
Typhoid Fever	..	..	..	..	..	..	..	..	..
Typhus (Flea-, Mite- and Tick-borne)	..	..	2	..	..	..	..	..	2
Typhus (Louse-borne)	..	..	..	..	..	..	..	..	..
Yellow Fever	..	..	..	..	..	..	..	..	..

[1] Figures in parentheses are those for the metropolitan area.

Committee in Medicine, 131 Macquarie Street, Sydney. Telegraphic address: "Postgrad Sydney." Telephone: BU 4497-8.

Post-Graduate Training Fellowships in Medicine.

The Post-Graduate Committee in Medicine has announced the names of the successful applicants who have been appointed by the Senate of the University of Sydney for two Post-Graduate Training Fellowships in Medicine. They are Dr. T. J. Claffey and Dr. David Howell, who are at present working in England. The Fellowships are for a period of twelve months.

At the Nuffield Department of Orthopædic Surgery and the Accident Service at Oxford, Dr. Claffey will carry out research in orthopædic surgery under the direction of Professor J. Trueta, particularly in regard to avascular necrosis of the head of the femur following fractures, with special reference to the site of the fracture in relation to the blood supply. The aim of the study will be to determine at the time of a fracture which types will inevitably lead to avascular necrosis.

Dr. Howell intends to study the use of radioactive isotopes in medicine, especially as applied to the field of obstetrics and gynæcology. His work will include basic training in the handling and control of radioactive material, which will be arranged through Professor R. S. Nyholm, D.Sc., Professor of Chemistry at the University of London, training in the principles of the use of isotopes in medicine at the special unit of the Medical Council established at the Post-Graduate Medical School of London, Hammersmith, and research work at the Department of Obstetrics and Gynæcology, Hammersmith, under the direction of Professor J. C. McClure Brown.

Nominations and Elections.

THE undermentioned have applied for election as members of the New South Wales Branch of the British Medical Association:

Ingle, Charles Bruce, M.B., B.S., 1955 (Univ. Sydney), 14 Cumberland Street, Cessnock, New South Wales.

Grima, Carmel Paul, registered in accordance with the provisions of Section 17 (1) (b) of the *Medical Practitioners Act*, 1938-1955, M.D., Malta, 1955, Bathurst District Hospital, Bathurst, New South Wales.

Brandson, Andrew Samuel, registered in accordance with the provisions of Section 17 (1) (c) of the *Medical Practitioners Act*, 1938-1955, 148 Ernest Street, Crow's Nest, New South Wales.

Feldman, Abram Isaak, registered in accordance with the provisions of Section 17 (1) (c) of the *Medical Practitioners Act*, 1938-1955, 22 Wallangra Road, Dover Heights, New South Wales.

Bryan, Keith, M.B., 1953 (Univ. Sydney), 231 Cleveland Street, Redfern, New South Wales.

Pohl, Hans Joachim, registered in accordance with the provisions of Section 17 (1) (c) of the *Medical Practitioners Act*, 1938-1955, 40 Rosebridge Avenue, Roseville, New South Wales.

Symons, Michael Frank, M.B., B.S., 1954 (Univ. Melbourne), 316 Burwood Road, Burwood, New South Wales.

Medical Appointments.

Dr. K. R. Shadwell has been appointed Government Medical Officer at Emerald, Queensland.

Deaths.

THE following deaths have been announced:

MEYERS.—Errol Solomon Meyers, on February 11, 1956, at Brisbane.

LOWSON.—James McKie Atkinson Lowson, on February 12, 1956, at Mansfield, Victoria.

Notice.

LAENNEC SOCIETY.

A CLINICAL MEETING of the Laennec Society will be held in the Scot Skirving Lecture Theatre, Royal Prince Alfred Hospital, Sydney, on Monday, March 5, 1956, at 8 p.m. Members and visitors are invited to attend.

Diary for the Month.

FEB. 25.—Tasmanian Branch, B.M.A.: Annual Meeting.
FEB. 27.—Federal Council, B.M.A. in Australia: Meeting at Hobart.
FEB. 28.—New South Wales Branch, B.M.A.: Ethics Committee.
MARCH 2.—Queensland Branch, B.M.A.: General Meeting.
MARCH 6.—New South Wales Branch, B.M.A.: Medical Politics Committee.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch (Medical Secretary, 135 Macquarie Street, Sydney): All contract practice appointments in New South Wales.

Queensland Branch (Honorary Secretary, B.M.A. House, 225 Wickham Terrace, Brisbane, B17): Bundaberg Medical Institute. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL or position outside Australia are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.

South Australian Branch (Honorary Secretary, 80 Brougham Place, North Adelaide): All contract practice appointments in South Australia.

Western Australian Branch (Honorary Secretary, 8 King's Park, West Perth): Norseman Hospital; all contract practice appointments in Western Australia. All government appointments with the exception of those of the Department of Public Health.

Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

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Members and subscribers are requested to notify the Manager, THE MEDICAL JOURNAL OF AUSTRALIA, Seamer Street, Glebe, New South Wales, without delay, of any irregularity in the delivery of this journal. The management cannot accept any responsibility or recognize any claim arising out of non-receipt of journals unless such notification is received within one month.

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